INHERITANCE OF RESISTANCE TO COMMON BACTERIAL BLIGHT (*XANTHOMONAS CAMPESTRIS* PV. *PHASEOLI*) DISEASE AND YIELD OF COMMON BEAN.

BY

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DECLARATION

I, BELARMINO AMADEU FAIFE DIVAGE, declare that the work presented in this thesis is my own research and has not been submitted for award of any degree in any University.

Signed……. Date……..

Belarmino Divage

This thesis has been submitted on our approval as supervisors

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DEDICATION

This work is dedicated to my parents Mr. And Mrs. Divage, who have foregone some pleasures of this world to see me through education and to my brothers and sister and my niece so that they may find inspiration to excel in academia.

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ACRONYMS AND ABBREVIATIONS

AUDPC : Area Under Disease Progress Curve

r-AUDPC : Relative Area Under Disease Progress Curce

CBB : Common bacterial blight

CIAT : International Center for Tropical Agriculture

PABRA : Pan African Bean Research Alliance

MAS : Marker Assisted Selection

QTL : Quantitative Trait Loci

SCAR : Sequence Characterized Amplified Region

SNP : Single Nucleotide Polymorphism

RAPD : Random amplified polymorphic DNA

Xcp : *Xanthomonas campestris pv. phaseoli Synonyms: Xap (Xanthomonas axonopodis pv. phaseoli), X. campestris pv. phaseoli var. fuscans (Xcpf)*

YCD : Yeast-extract-dextrose-calcium-carbonate agar

ABSTRACT

The common bean (*Phaseolus vulgaris* L.) is a legume grain crop with great importance in East Africa, being a source of food and income for most rural households. In Uganda, common bean is a major source of food security being a readily available and popular food to both the urban and rural population. However, productivity of the crop is limited by many diseases, common bacterial blight (CBB) caused by *Xanthomonas campestris* pv *phaseoli* (Xcp) being one of them. The use of natural resistance to CBB is the most effective and environmentally sound approach among others to control this disease. This study aimed at i) identifying new resistance genes to CBB and ii) understanding the mode of inheritance in the newly identified sources of resistance. Eighty genotypes (65 lines from the PABRA regional nutritional nursery, 10 interspecific lines (*P. coccineus* x *P. vulgaris*) coded ALB, five documented sources of CBB resistances coded VAX and 24 newly developed resistance lines coded ACC were screened for CBB resistance in a screen house at CIAT Uganda. A *Fuscans (Xcpf)* variant of *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) isolate named *"Kawempe 1"* was used. From the screening, six genotypes namely JESCA, RWV 2070, RWR 2154, MIB 456, NUA 45 and MCM 2001 were found to have good resistance to CBB. To determine the mode of inheritance of the identified resistance in these six genotypes, crosses with locally prefered genotypes were made, two landraces; Masindi Yellow (large seeded yellow) and Kanyebwa (medium sized red speckled sugar bean), and two released varieties; K131 (small seeded carioca seed type) and K132 (large seeded red mottled) using North Carolina Design II matting design**.** All the F1 seed available was planted and F1 progenies were advanced to F2 generations in screen house and evaluated for resistance to Xcp using *Xanthomonas campestris* pv. *phaseoli* var.*Fuscans (Xcpf)* variant of *Xanthomonas Campestris* pv. *phaseoli* (Xcp) "Kawempe 1" .

Heritabilities estimates and segregations patterns showed that additive effects predominated over non additive ones with quantitative inheritance. Among the six resistant MCM 2001 and RWV 2070 showed the strongest GCA effect hence most effective resistance. Masindi Yellow X RWR 2070, Kanyebwa x RWR 2070, K 132 x MIB 465, K 131 x JESCA and K 131 x MCM 2001, were considered the most desirable crosses for CBB resistance. The crosses K132 x

JESCA, K132 x MIB 456 and Masindi Yellow x RWR 2070 had good mean values for all the yield parameters under analysis. Chi-square tests for goodness of fit showed the presence of more than one gene controlling the resistance to CBB on the materials used on this study. Some of the susceptible parents (K132 and Kanyebwa) were shown to posses factors contributing to CBB resistance. The heritability estimates for broad-sence and narrow sence coefficient of genetic determination was 0.65 for both, because the SCA variance was negligible due to its negative value, the Bakers ratio was 1, with more than one gene involved with epistatic interaction.

From the study findings, it is recommended that the characteristics of resistance to CBB should be investigated in every new parental source when they are initially introduced into the breeding programme. It is also recommended that breeding methods such as crossing and selfing or backcrossing that make the best use of additive variance, should be used to transfer CBB resistance into susceptible commercial and preferred varieties, since the additive gene actions were more important than non-additive gene effects. Generations could also be advanced by the single-seed-descent method or F2-derived families harvested in bulk, due to high hereditability estimation value. Later the promising genotypes should be subjected to multi-location trials to test the stability of their performance while enriching findings regarding their gene action. The promising stable varieties identified should be subjected to selection, preferably with participation of farme

CHAPTER ONE

INTRODUCTION

1.1 Background

Common bean (*Phaseolus vulgaris* L.) is one of the most important food legumes consumed worldwide, especially in developing countries (Durham**,** 2011). Common bean is a great source of minerals (Ca, Cu, Fe, Mg, Mn, Zn) and protein for human diets (Broughton *et al.,* 2003). It is also an important source of dietary fibres (Gepts *et al.,* 2008).They are also a relatively good source of water soluble vitamins especially thiamine (0.9-1.2 mg/100gm), riboflavin (0.14-0.27 mg/100gm), niacin (1.16-2.68 mg/100gm), folic acid (0.17 mg/100gm) and vitamin B6 (Mazza, 1998). Worldwide, about 12 million tons of beans are produced annually (Broughton *et al.,* 2003). Latin America produces about 5.5 million tons of beans annually, and is the highest producing region in the world; with Brazil and Mexico being the largest producing countries. Africa is the second most important common beans producing region with an annually output of about 2.5 million tons. The major producers of common beans in this region are Tanzania, Uganda, Kenya, Rwanda, Burundi and the Democratic Republic of Congo (DRC) (Broughton *et al.,* 2003).

In Uganda, beans plays an important role for both rural and urban population for food security, providing 25% of the total dietary calorie intake and 45% of the protein intake, with per capita consumption estimated to be 19 kg/year. They are also a major source of complex carbohydrates, essential micronutrients, dietary fibre, vitamin B and antioxidants (Trust, 2012). For a long time in Uganda, beans have been produced mainly for food security at household level, but currently beans production is a major source of income (Trust, 2012).

Beans in Uganda are grown in two seasons (March to June, and September to November), with high yields recorded in the second season (September to November) due to the relatively high amount of rainfall (MAAIF, 2010). The current bean production in the country is estimated at 425,400 metric tones (FAO, 2014). The major beans varieties grown in Uganda are K132,

Nambale short, Kanyebwa and Masindi yellow (Trust, 2012). recording the lowest (0.9 ton/ ha) (MAAIF, 2010). However, depending on location and environmental factors, farm yields for most of the varieties range between 0.4 - 0.7 ton/ha, generally low compared to the research station yields of 1.5 – 2.5 ton/ha (MAAIF, 2010). Uganda exports about 20% of the beans produced, suggesting that most of the beans are consumed locally (UEPB, 2005). In 2011 about 28,055 ton of dried beans were exported. In the recent past the highest export of 41,141 MT were recorded in 2009 (ITC, 2012). The main destinations for these exports are Kenya, South Sudan, Democratic Republic of Congo, Tanzania, Burundi, UK and USA.

Globally, common bean production is affected by several factors. These factors are biotic, abiotic and socio-economic. Among the biotic factores are fungal, viral and bacterial diseases (Ferreira *et al.*, 2003). In the past few decades, soil borne deseases such as root rots have emerged as a greater problem especially those caused by *Pythium spp* and *Fusarium spp*. (Beebe *et al.,* 2012). Intense cultivation under increasing population pressure, without fallow periods or adequate crop rotation, results in declining soil fertility or soil compaction, or both, and in build-up of pathogen inoculum in the soil (Wortmann *et al.,* 1998). Soil compaction inhibits root growth and the potential for plant recovery after infection (CIAT, 2006).

Socio-economic condictions affecting common beans production include limited availability of breeder and foundation seeds and low participation of private traders in the input distribution system (UBS, 1999). High transportation costs partly caused by poor roads, poor communication and storage infrastructures are among the other socio-economic factors affecting bean production in Uganda (MAAIF and MFEPD, 2000).

Bacterial diseases are particularly important in both tropical and temperate regions where beans are produced and cause significant yield loss (de Jesus *et al.,* 2001). Common bacterial blight (CBB), caused by *Xanthomonas campestris* pv. *phaseoli* (*Xcp*), is a major seed-borne disease of common bean worldwide (Tar'an *et al*., 2001). Common bacterial blight symptoms are commonly first seen as small, light green, water-soaked or translucent spots (lesions) on the leaves. When conditions are warm and wet, lesions enlarge rapidly and even merge. In highly susceptible varieties, lesions continue to expand and as such leaves soon become ragged and torn

by wind and rain. Later, they wither and drop off (Harveson, 2009). Pod infection by CBB often causes discoloration, shriveling and bacterial contamination of seeds (Schwartz, 2011). Most common bean yield loss due to CBB is attributed to premature defoliation from leaf lesions (Goodwin, 1992).

Bean yield loss due to *Xcp* depends on the intensity of the disease and prevailing environmental conditions and susceptibility of the cultivars (Asensio *et al*., 2006). In Africa, CBB is ranked the fourth most important bean disease. It has been reported as a major disease especially in Uganda, Zambia, Zimbabwe and South Africa (Fourie, 2002, Zamani *et al.*, 2011). Total losses caused by CBB in Africa can reach about to 220 000 t / year, of which 146 000 t is lost in East Africa and while almost 70.000 tons per year is lost in South Africa (Wortmann *et al.*, 1998, Opio *et al.*, 2002).

1.2 Statement of the Problem

In Uganda and other East African countries, lack of resistance to common bacterial blight in popularly grown varieties and poor seed health are the major factors promoting commom bacterial blight disease epidemics. Use of farmer saved seed is a common practice in the region, further exercabating the situation, as CBB is a seed borne disease (Opio *et al.*,1993). Even though the disease is recognised as important to bean production in Uganda, little efforts have been directed at addressing it. Use of pesticides to control CBB have proven unsatisfactory (Osdhagi *et al.*, 2009) as has seed treatment with antibiotics or foliar application of other chemical products. These are costly, but also have long-term implications on the health of plants and animals (Forbes and Bretag, 1991; Fininsa, 2003). Some cultural practices such as crop rotation and weed control have been reported to reduce CBB disease incidence. However, use of clean, pathogen-free seed combined with host plant resistance remain the most practical, effective and environmentally-sound approaches to control the disease (Zanatta *et al*., 2007; Shi *et al*., 2011).

In Uganda, two common bean genotypes MCM 5001 and XAN 112 were reported to be resistant to five *Xcp* strains (Opio *et al.*, 1993). These have never been released due to lack of important farmer preferred attributes. The linkage of resistance with undesirable traits (Liu *et al*., 2008),

different genes conditioning resistance in different plant organs such as on leaves, pods, and seeds (Liu *et al*., 2009; Mutlu *et al*.,2008; López *et al*., 2006; Mkandawire *et al*., 2004), make breeding for resistance to CBB on beans complex. Further, inheritance of CBB resistance in beans is also not well understood. Some scientists have reported resistance to CBB is quantatively inherited (Kelly *et al*., 2003; Miklas *et al*., 2006). Breeding programmes in Uganda lack sources of CBB resistance in varieties that are adapted to local environmental conditions. This as resulted in little progress towards breeding for CBB resistance in beans. Despite all this, it is important that efforts be made to circumvent these barriers and introduce CBB resistence into locally adapted varieties. Resistance sources to CBB have however been identified in some common bean (*P. Vulgaris*) lines and the wild relative *P. acutifolius* and *P.coccineus* (Tar'an *et al.*, 2001). Resistance in these sources has even been successfully transferred into some common bean lines (Singh and Munoz, 1999; Osdaghi *et al.*, 2009). However, most of developed resistant germplasm lacks farmer preferred traits for them to be release and easily adopted, by farmers. Therefore, suseptible varieties continue to dominate. In Uganda, efforts to improve resistance to CBB in beans more made targeting locally acceptable varieties Kanyebwa and K20 more than ten years ago. The sources of resisistance used included PI 207262, lAPAR 16, BAC 6, GN Jules' GN Nebrasca selection 27, XAN 112 and XAN 159, all of which more exotic (Opio and Namayanja, 2002).Unfotunatilly, not much progress was made as the materials developed lacked desirable attributes for farmer adoption (Opio and Namayanja, 2002). It is therefore envisoned that using locally adapted sources of resistance would most likely result in higher chances of developing CBB resistant varieties with farmer preferred attributes. Unfortunatitly, no comprehensive screening of locally adapted cultivars has been made in Uganda. It is also important that for each source of identified source of resistance, the mode of ist inheritance in selected locally adapted varieties be ilucidated. It is known that the mode of CBB resistance, inheritance is diffent for many cultivars. This study therefore aimed at identifying and characterizing local sources of effective resistance genes to common bacterial blight disease of common bean in Uganda and determining the mode of inheritance of this resistance.

1.3 Justification

Available literature shows clearly that resistance to CBB is generally a complex trait, reported to be quantatively inherited (Kelly *et al.* 2003; Miklas *et al*. 2006). The linkage of resistance with undesirable traits (Liu et al. 2008), different genes conditioning resistance in different plant organs such as leaves, pods, and seeds (Liu *et al.*, 2009; Mutlu *et al.*, 2008; López *et al.*, 2006; Mkandawire *et al.*, 2004). Further more is important to understand inheritance to CBB better. Identifiying and using locally adapted sources of resistance and understanding their genetic architecture, would probably result in higher changes of developing CBB resistant varieties that have farmer preferred attributes. Unless souces of resistance among locally adapted varieties is found and the inheritance of CBB resistance associated with each are determined, success in introducing resistance into locally adapted and farmer preferred cultivars will remain distant. This implies that disease will remain rampant. The population in Uganda will therefore not benefit from the numerous uses of beans.

1.4. Research objectives

The general objective of this study was to contribute to common bean improvement through identification of sources of resistance to common bacterial blight disease and improved approaches to breeding.

Specific objectives:

1. To characterize diverse common beans lines for their resistance to CBB infestation
2. To determine the mode of inheritance to CBB resistance in locally adapted sources.

Research Hypothesis

This research tested the hypotheses that:

1. There is resistance to CBB within locally adapted cultivars as good as that in exotics lines.

1. The gene action governing the inheritance of resistance CBB is additive in nature.

CHAPTER TWO

LITERATURE REVIEW

2.1. Origin of common bean

According to the evidence provided by archaeological observations dating from 10000 to 8000 BC, the common bean has its origins in the Americas (Diniz and Távora, 2009). Originally domesticated in the regions of south America, central America and Mexico, common beans have since then expanded into other regions of America (from 35° S to >50 N latitude and altitude of 3000m (Gepts and Bliss, 1990). Beans were later introduced in Africa, Europe, Asia and Oceania (Gepts and Bliss, 1990; Singh and Miklas, 2007). Through the process of domestication, common bean has been modified in several morphological and physiological aspects such as from indeterminate climbing to determinate bush type, sensitivity to insensitivity to long photoperiod, from small to large leaves, pods and seeds among others (Singh and Miklas, 2007).

2.2. Agronomic characteristics of Common Beans

Beans are classified according to their morphological and physiological characteristics. Depending on the growth stages, bean development is classified into vegetative (V0-V4) and reproductive (R5-R9). According to growth habit, beans can be described as being determinate (I) or indeterminate (II-VI) (Pastor-Corrales *et al*., 1987). Generally, common bean is considered a short period crop taking about 65-110 days to physiological maturity (Buruchara, 2007). However, this time can extend up to 200 days after planting for climbers when grown in the high cool elevations (Graham and Ranalli, 1997; Gomez, 2004). The crop is not very demanding in terms of soil conditions provided it is reasonably fertile, well drained and has no interference with regard to germination and emergence (Wortmann *et al.,* 1998). In terms of precipitation, common beans require moderate amounts of water (300-600 mm), ensuring adequate amounts during and immediately after flowering (Katungi *et al.,* 2009).

2.3. Importance of common beans

Common beans (*Phaseolus vulgaris*) is a legumenous crop and traditionally a basic food crop in many developing countries. It is a major plant protein source in rural and urban areas (Abo-Elyousr, 2006). Beans can be consumed in many diffent ways. They can be as mature grain or as immature seed. Its leaves and pods are also consumed as a vegetable (Broughton *et al.*, 2003). Common bean is low in fat and is cholesterol free; a regular diet with beans brings great benefits to human health because it reduces the risk of developing cancer, diabetes and heart disease (Matella, 2006). Common beans is a rich source of zinc and iron. It is thus very good for people infected with AIDS, because these two micronutrients are commonly depleted from such people (Buys, 2002). Therefore, diets containing beans are recommended to HIV-infected patients (ADA, 2004; Kruzich *et al.*, 2004).

The common bean also has high economic potential. It is a source of income through sale of fresh pods and dry seeds (Wortmann, 1998; Broughton *et al.*, 2003). World-wide, the common bean has an annual production value estimated at over US $ 11 billion (USDBC, 2014). In Africa, the common bean is a significant and growing source of income for many rural households, with annual sales value of over $580 million (PABRA, 2008).
In addition, common bean plays an important role in improving soil fertility. It is therefore important in low input farming systems common in many parts of Africa (Katungi, 2009).

2.4. Common beans production in Uganda

In Uganda, beans are an important source of food security, primarily because it is cheap and readily accessible to both the urban and rural population (Mauyo *et al.*, 2007)**.** The common bean is produced in all of the eight main agro-ecological areas In the country (Wortmann and Allen, 1994). On average, the size of bean gardens in Uganda is about 0.3 ha per household. About a third of the grain produced is sold while another third is consumed. The rest is stored or used for other purposes, (UBOS, 2010). The current bean production in Uganda is estimated at 425,400 metric tones (FAO, 2012), with average from level yields of 556.2 kg/ha.

2.5. Constraints to common beans production in Uganda

Common beans production in Uganda is constrained by several biotic and abiotic factors (Wortmann *et al*., 1998; CIAT, 2008). The biotic factors affecting bean production in the country are insect pests and diseases. Among the abiotic factors, drought and low soil fertility due to nitrogen, phosphorus, and zinc deficiencies, as well as toxicities due to aluminum and manganese are among the major constraints in beans production in the country (Singh, 2001). The most important insect pests of bean are bean fly (*Ophiomyia phaseoli* Tryon), foliage beetle (*Ootheca* sp*;* Coleopteran: Chrysyomelidae) black aphid (*Aphis fabae*) and flower thrips (*Megalurothrips sjostedti;* Thysanoptera: Thripidae) (Katwijukye and Kyamanywa, 1997).

The important diseases include common bacterial blight (CBB) (,*Xanthomonas campestris* pv. *phaseoli Xcp*) halo blight [*Pseudomonas syringae* pv. *phaseolicola* (Burkh.)] and bacterial brown spot (*Pseudomonas syringae* pv. *syringae* van Hall) (Namayanja *et al.*, 2007; Nkalubo *et al.*, 2009).

Bean common mosaic virus (BCMV) and bean golden mosaic virus (BGMV) are among the most important bean diseases caused by viruses. Some of the important bean diseases are caused by fungal pathogens. These include the root rot complex (*Fusarium solani*, *Pythium* spp. and *Rhizoctonia solani*.), angular leaf spot [*Phaeoisariopsis griseola* (Sacc.) Ferr.] and anthracnose [*Colletotrichum lindemuthianum* (Sacc. and Magnus) Lams.-Scrib.] (Opio *et al.*, 2001; Namayanja *et al.*, 2007; Nkalubo *et al.*, 2009). Common bacterial blight (CBB) caused by *Xanthomonas campestris* pv. *phaseoli* is ranked the fourth most important bean disease in Africa causing an estimated loss of 220,000 t/year (Wortmann *et al*., 1998). Of these losses, 66% is reported to occur in Eastern Africa and nearly 32% in Southern Africa (Wortmann *et al.*, 1998). The disease has been reported to be of major importance in Kenya (Njungunah *et al*., 1981), Malawi (Edje *et al*., 1981), Uganda, Burundi (Opio *et al*., 1993) and Tanzania (Karel *et al*., 1981). Yield loss in Uganda due to common bacterial blight is reported to range between 10 to 40% and is more prevalent and severe in the low altitude areas of Uganda (Opio *et al*., 1996).

The disease is favoured by high temperature, rainfall and humidity. Recent reports indicate that East Africa is increasingly becoming wetter making it more conducive for CBB disease development (Buruchara *et al*, 2011).

2.6. Epidemiology of common bacterial blight

Common bacterial blight is endemic in almost all bean growing areas especially in hot lowland and mid altitude areas (Mkandawire *et al.,* 2004). Generally common bacterial blight is favored by conditions of high humidity and temperature (CIAT, 1989; Karavina *et al.,* 2011; Akhavan *et al.,* 2013). The disease causes greater damage to plants growing at 28-32^{o}C (Macnab *et al*., 1983). The source of CBB inoculum is internally or externally *Xcp* contaminated seed. In some cases contaminated or injected seed appears symptomless (Jung and Skroch, 1997; Darrasse, 2007). Other inoculum sources for common bacterial blight are contaminated bean debris and weeds which act as alternate hosts of the pathogen (Zamani *et al.*, 2011).

Dissemination of bacterial blight bacteria can be viewed as long distance and short distance. Short distance pathogen dissimination includes in-field and plant-plant tramiossion. In-field transmission is facilitated by wind-driven rain. Insects such as whiteflies, leaf miners and beetles are reported to facilitate in-field plant to plant transmission (Zhang *et al.*, 2002). People and contaminated equipment are also considered short distance disease vectors (Gilbertson and Maxwell 1992; Harveson, 2009). For long distance transmission, the most important means is considered to be seed (Gilbertson *et al*., 1990). *Xcp* can survive in seed, crop debris and weeds for up to 10 years (Boyle *et al.*, 2007; Zhang *et al.,* 2002). Prolonged survival of up to thirty six years has also been reported (Fourie, 2002).

Cells of *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) enter bean plants through openings such as stomata in leaves and other plant organs and through hydathodes at leaf margins. Wounds on plants such as those created by wind-blown soil particles are also major entry points for bacteria (Rudolph,1993; Fourie, 2002). After entry, bacteria invade intercellular spaces, causing gradual dissolution of the middle lamella (Karavina *et al*., 2011). Presence of sufficient numbers of

bacteria in the vascular system especially in the xylem tissue may cause plant wilting by plugging the vessels or disintegration of cells walls (Yoshii, 1979).

2.7. Symptoms of common bacterial blight on common bean

Common bacterial blight (CBB) caused by *Xanthomonas campestis* pv. *phaseoli* and its fuscans variant, *Xantomonas campestris* pv. *phaseoli* var. *fuscans.* These are geniticaly disctint but cause the same symptoms on the host plant (Fourie and Herselman, 2011). Bean bacterial blight is consequently categorised as common or fuscous. Common blight caused by the fuscans variant is known as fuscous blight of beans (Cabi, 2014). Symptoms of both of these are commonly first seen as small, angular, light green, water-soaked or translucent lesions on the leaves.

Plate 1: Sypmtoms of common bacterial Blight on leaves and pods (Source: University of Illinois at Urbana-Champaign, 2000).

During warm, wet conditions lesions rapidly enlarge and merge. In the leaves and stems lesions can be found at the margin and in interveinal areas of the leave. Infected regions appear flaccid and are encircled by a narrow zone of lemon yellow tissue which later turns brown and necrotic. Serious infections may cause defoliation or stem girdling (Karavina *et al.,* 2011). Pod lesions start as round, water-soaked dots that enlarge, merge, dry, and form sunken, irregular, frequently reddish brown blotches. When infection is severe entire pods may be badly shriveled and die (Harveson, 2009). Symptoms on white or light-coloured seeds are evident as butter-yellow or brown spots distributed throughout the seed coat or restricted to the hilum area (Mabagala, 1997).

If infection occurs during pod and seed development, infected seed may rot or shrivel or may be wrinkled (Karavina *et al.*, 2011).

2.8. The Pathogen- Xanthomonas campestris pv phaseoli (Xcp)

2.8.1. Taxonomy and morphology

Taxonomically the species *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) belongs to the genus *Xanthomonas* within the family *Xanthomonadacea* and order *Xanthomonadales* (Benson, 2009). The genus *Xanthomonas* is notable by its phytopathogenic and phenotypic diversity, a factor that has created immense challenges in its taxonomy and classification (Vauterin *et al.*, 2000).

The genus *Xanthomonas* comprises about 27 species that can cause disease in approximately 400 host plants and pathogenic strains show a high degree of host specificity (Ryan *et al.*, 2011). With intent to better explain the taxonomy and the differences between species belonging to this genus, many studies using different techniques are still on going to establish a criterion for classification of this pathogen (Durham, 2011). *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* is its variant, despite the fact that *Xanthomonas campestris* pv. *phaseoli* and *X. campestris* pv. *phaseoli* var. *fuscans* present almost the same pattern of symptoms in the host, they are genetically distinct with no geographical differentiation (Mahuku *et al.*, 2006).

Plate 2. Colonies bacteria of *Xanthomonas campestris* pv. *phaseoli* (*Xcp*) growing in media. Source: CIAT Laboratories,Uganda-Kawanda, 2014.

Morphologicaly, the bacterium is characterized by individual cells that are straight rods (0.4-0.7 x 0.7-1.8µm) with a polar flagellum. The bacteria grows on several media such as agar, Yeast Dextrose Agar and MXP producing characteristic yellow, mucoid colonies. The pathogen is also characterised by glistering and convex colonies with, entire margins or surrounded by zones of starch hydrolysis (Schaad, 1988; Mabagala and Saettler, 1992).

2.8.2. Pathogen survival and disease development

Bacterial pathogens can survive in previously infected bean stubble (straw and seed) as well as in previously infested soils, bacteria can survive at temperature between 5°C and 39°C (Dye and Lelliot, 1974). It can also survive in both epiphytic and endophytic forms.(Weller and Saettler, 1980). It is the epiphytic population which has a very important influence on disease development and subsequent epidemics (Beattie and Lindow, 1999). This asymptomatic period may lead to such a large bacterial population from which disease develops later if more favorable environmental conditions occur (Wilson *et al.*, 1999). The following year, the surviving bacteria can multiply in emerging plants if environmental conditions are favorable. The seed is considered as the main and primarily source of *Xcp* in common bean (Weller and Saettler, 1980; Jung *et al*.,1997; Schwartz *et al.*, 2005).

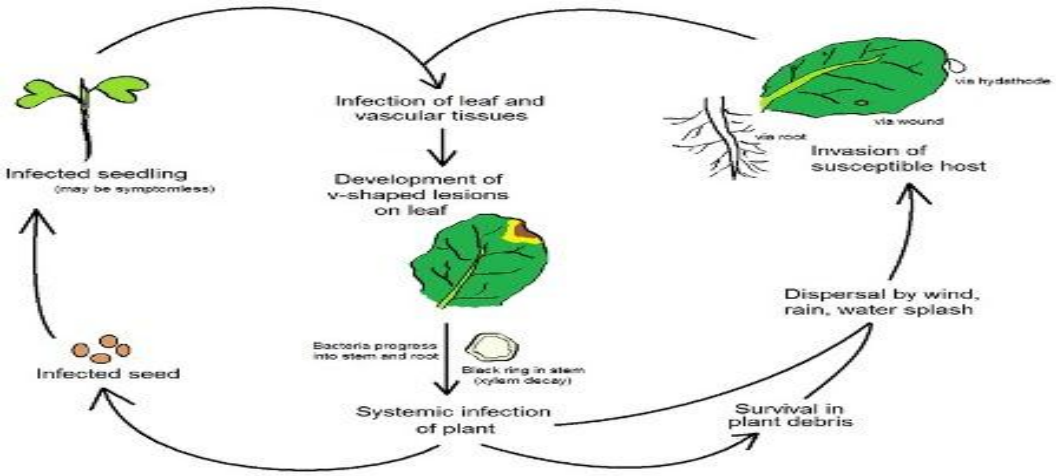


Plate 3. Life cycle of *Xanthomonas campestris* **pv**. *phaseoli* (Kwan, .2010)

It can also be spread from plant to plant and field to field in many ways including contaminated debris by wind, direct contact, animals, tools, human, rain and irrigation water (Schwartz *et al.*, 2005). *Xcp* penetrate bean plants through openings such as stomata in the leaves and other plant parts and through hydathodes on the leaf margins. Wounds created by wind blown soil particles also permit entry of bacteria (Rudolph, 1993, Akhavan *et al.,* 2013).

Bacterial penetration into plant tissue is favoured by temperatures ranging from 25-35°C and high rainfall and high humidity (Agrios, 2005; Schwartz *et al.*, 2005). Within cells of the host plant, bacteria begin to multiply, then rupture the membrane, digest these cells, thereby leaving large lesions characteristic of the disease (Agrios, 2005; Schwartz *et al.,* 2005). In the vascular system bacteria also quickly spreads to other parts of the plant and eventually infects seed (Swings and Civerolo, 1993; Durham, 2011).

2.9. Breeding for resistance to common bacterial blight on common beans

Resistance to CBB in common bean may confer physiological mechanisms to reduce or even stop the circulation of bacteria in plant tissues, therefore reducing the accumulation of bacteria attacking the leaves or internal tissues in seeds (Goodwin *et al.,* 1995; Aggour and Coyne, 1989). The genus *Phaseolus* has three principal gene pools. The primary gene pool is represented by *P. vulgaris*, the secondary is represented by *P. coccineus, P. costaricenis* and *P. polyanthus* while the tertiary is represented by *P. acutifolius* and *P. parvifolius.*

These are the three gene-pools that have provided sources of resistance to CBB (Singh and Schwartz, 2010; Singh, 1999). However, for successful crosses between the primary and tertiary gene-pools, embryo rescue is necessary. Crosses between the primary and secondary gene pools do not require embryo rescue (Singh and Schwartz, 2010; Parker and Michaels, 1986). Unfortunately the level of resistance to CBB in *P. vulgaris* (which is the cultivated bean) is limited and thus wide crossing is inevitable. The highest level of resistance to CBB has been reported in tepary bean (Singh and Munoz, 1999; Zapata *et al.,* 1985), followed by *P. coccineus* (Scarlet runner) (Singh *et al.,* 2001; Singh and Munoz, 1999; Miklas *et al.,* 2006).

Resistance genes for CBB have been successfully transferred to common bean from these sources (Singh and Munoz, 1999). Important interspecific crossings between tepary beans and *P. vulgaris* were made to develop different lines and cultivars with resistance to CBB in bean. Some of these lines include OAC 88-1 (Scott and Michaels, 1992), VAX1 and VAX 2 (Munoz and Singh, 1999) XAN 159, XAN 160 and XAN 161 (Beebe *et al.*, 1981; McElroy., 1985). Regardless, low to moderate levels of resistance were introgressed from *P. coccineus*. (Durham, 2011). XAN 159 was subsequently used to introduce resistance into two lines HR45 and HR67 (Park and Dhanvantari, 1994; Park *et al.,* 2006).

Dhanvantari and Park (1987) have reported four CBB resistance lines, C1, C2, C3 and C4 that resulted from inter-specific crosses between common bean and *P. Coccineus.* Miklas *et al.* (1994) in Mexico released Tars VCI-4B line which is a source for resistance to multiple diseases including CBB. This line was developed from two *P. coccineus* lines, PI311950 and PI311977. Also four other CBB resistant bean lines, ICB-3, ICB-6, ICB-8 and ICB-10 were released by Miklas *et al.,* (1999) which derived their CBB resistance either from *P. coccineus* or from Great Northern varieties. The great northern cultivars GN# 1 and GN#1 Selection 27 (GN#1Sel 27) were derived from common bean cultivar, Montana No. 5. The only other example of high levels of CBB resistance derived from *P. vulgaris* has been reported in the landrace which was released in 1947 from a selection out of the common great northern landrace (Miklas *et al.,* 2003)

CBB Resistance was also transferred from tepary bean into *P. vulgaris* via hybridization between PI440795 (*P. acutifolius*) and 'ICA Pijao' (*P. vulgaris*) and the F1 progeny crossed with 'Ex Rico 23' (*P. vulgaris*). PI 440795 is the source of CBB resistance in OAC-88-1 and OAC- Rex line and cultivar respectively. Using embryo rescue, inter-specific crosses between the common bean cultivar ICA Pijao and the tepary bean accession G40001 resulted in VAX's lines VAX1 to VAX 6 (Mejia-Jimenez *et al*., 1994; Singh and Munoz, 1999). Recently a dominant gene conferring resistance to CBB was found in the small white bean line PR0313-58 (Zapata *et al*., 2010).

2.10. Mode of inheritance and gene action of common bacterial blight resistance in beans

Studies conducted so far show that the mode of inheritance of CBB resistance is complicated. Miklas *et al*. (2006) and Chataika *et al*. (2011) reported that the mechanism of inheritance of CBB resistance in beans is quantitatively inherited with major gene effects. On the other hand, Tar 'an *et al*. (2001) reported that CBB resistance was conditioned by approximately one to five genes with additive action and that it may be influenced by plant architecture, growth habit and maturity period. According to Chataika *et al.* (2011), climbing beans with their vigorous vegetative growth and spreading canopy and bush with their canopy crowded close to the ground level creat different microclimates and influence performance of resistance genes. According to Singh (1991), the nature of inheritance to CBB also depends on the genotype used as the susceptible parent and also the source of resistance and that it can be determined by both major and minor genes.

Supported with molecular markers, studies have identified at least 22 QTL's for resistance to CBB present along all the 11 chromosomes in different bean lines. It was also established that these QTL are influenced by environmental conditions, genetics, disease pressure and certain agronomic characteristics (Miklas *et al.*, 2006). Negative epistatic interactions between QTL for CBB resistance was also on common beans (Vandemark *et al.*, 2009). Including negative associations between agronomic traits and resistance QTL (O'Boyle *et al.*, 2007). That complex nature of resistance to CBB and its major environmental influence on the symptoms characteristic development make challeging to move forward to improve strategies for CBB resistance (Osdaghi *et al*, 2009).

CHAPTER THREE

IDENTIFICATION OF NEW SOURCES OF RESISTANCE TO COMMON BACTERIAL BLIGHT IN BEANS IN UGANDA

3.1. Introduction

Host plant resistance remains the most effective and environmentally-sound approach to control CBB (Shi *et al.*, 2011). Therefore, it is important to identify sources of resistance to *Xcp* to facilitate breeding programmes (Osdaghi *et al.*, 2009). In Uganda most of the commercial and farmers preferred culticars lack resistance to CBB. Since the identification of CBB as an important disease in Uganda in 1960 (Leakey, 1963), no particular attention was given to it until 1983. From 1986 - 1994 considerable effort was devoted on pathology and breeding (Opio and Namayanja, 2002). The breeding work was mainly focused on improving locally acceptable varieties (such as Kanyebwa, K20) for resistance to CBB using different exotic materials as donor parents. The promising lines generated from these crosses were incorporated in the CBB nursery that was distributed to some countries in Africa, but because they lacked some desired attributes, these lines have never been released in Uganda. Therefore prevailing the use of susceptible cultivars, conditioning yield and seed quality as well. This study aimed at identifying new sources of resistance to *Xcp* and to identify possible CBB resistant parents for use in improving locally adapted common bean varieties in Uganda.

3.2. Materials and methods

3.2.1. Location of the study

The study was conducted at the International Centre for Tropical Agriculture (CIAT) based at the National Agricultural Research Laboratories (NARL) Uganda-Kawanda. NARL - Kawanda is located (0°25°N,32°32°E), 13km North of Kampala and at 1195m above sea level. Day length at Kawanda is 12 h throughout the year. Average daily temperatures for Kawanda are 15.8^{o}C minimum and 29.8^{o}C maximum with mean relative humidity of 76%.

3.2.2. Description of the genetic material

Two sets of nurseries were evaluated for resistance to CBB in this study. The first set of material comprised of 80 genotypes. Of these, 65 were lines from the PABRA regional nutrition nursery. This nursery is being evaluated regionally in Africa as high Fe and Zn content candidate lines. Another ten (10) lines (coded ALB) were selections from interspecific crosses between *P. vulgaris* and *P. coccinueus* selected for Aluminum tolerance and multiple disease resistance. The other 5 lines (the VAX lines) were selected from lines specifically developed for CBB resistance from an interspecific cross between *P. vulgaris* and *P. acutifolius* (Mejia-Jimenez *et al.,* 1994; Singh and Munoz, 1999). The VAX lines have been used in different breeding programs worldwide as a source of CBB resistance (Fourie, 2011).

The second set included 24 ACC lines recently developed for CBB resistance at the CIAT Headquarters in Colombia. Also included in this study were varieties K131 (NABE 2) and K132 (CAL96) released in Uganda in 1994. These varieties are highly marketable, but susceptible to many production constraints (David Kirkb *et al.,* 1999) including CBB. Two other local landraces Kanyebwa and Masindi Yellow were included in the study as checks. In terms of seed size, the above germplasm included 58 small-seeded, 30 medium seeded and 21 large seeded. Considering growth habit; 57 were bush types while 52 were climbers (Table 1).

Table 1. Germplasm evaluated for resistance to common bacterial blight

Nursery	No lines	Description	Sources
1. Regional nutrition nursery	65	Fe and Zn content	CIAT-Uganda/ PABRA
2. VAX lines	5	CBB resistant parents	CIAT-Colombia
3. ALB lines	10	Interspecific lines (P. *Coccineus*) bred fo Al^{2+} tolerance	CIAT-Uganda/PABRA
4. ACC lines	24	CBB resistance	CIAT-Colombia
5. Officially released	2	Popularly grown varities in Uganda (K131, K132)	CIAT-Uganda/ PABRA

	varieties				
6.	**Local land races**	2	Kanyebwa and Masindi yellow popularly grown		CIAT-Uganda/ PABRA
Total		**108**			

3.2.4. Planting and experimental design

Seed of these genotypes were planted in five litre volume plastic pots containing a potting mixture of forest black soil, lake sand and decomposed farm yard manure in a ratio of 3:1:1. NPK (17:17:17), fertilizer was added to these soil. To apply fertilizer, hundred grams (300 gm) of NPK were diluted in 10 litres of water, from which 100 ml were put in the potting mixture on a weekly basis until the reproductive stage of pod filling (R8). The set of 80 lines were planted in an alpha lattice design with two replications, eight blocks with 10 lines/block. The second set of 24 ACC lines were planted in a randomized complete block design with two replicates.

3.2.5. Resistance confirmation and isolate selection

To confirm the results, a experiment was conducted were the selected six genotypes were screened using six different CBB isolates namely CBB 1, Kawenpe 1, KIS-wa-001, Kyanga, MA-F-011 and MSD-B-05, from which also the virulence of the "*Kawenpe 1*" isolate, was compared with other five above mentioned CBB isolates. The varieties Kanyebwa and Masindi yellow were used as susceptible checks and three VAX's lines (VAX1, VAX2, and VAX3) were used as resistant checks. The inoculation was done at 17 days after planting using the razor blade technique and severity was assessed at 10, 14, 35 and 56 DAI (days after inoculation) using a scale 1 to 9 (CIAT, 1987); where: 1= is reserved for an absence of symptoms which is often equated with highest level of resistance and 9 = represents extreme susceptibility (presence of severe symptoms, damage, or stress). Finally scores were used to group test lines into resistance categories. These categories were: Resistant (1-3), intermediate (4-6) and susceptible (7-9).

3.2.6. Inoculum multiplication and inoculation of genotypes

The isolate used in this screening was "*Kawempe1"* from CIAT-Uganda, which is a *fuscous* variant of *Xanthomonas campestris* pv. *phaseoli (Xcp)* (Mutlu *et al.*, 2008). A stored culture of *Xcp* isolate "*Kawempe 1"* was revived by growing it on Yeast Dextrose Carbonate Agar (YDCA at 28°C for 48hrs. After 48 hrs, three to five ml of distilled water was added to culture plates and bacteria scrapped off the media and mixed with water using a sterilized bent-glass rod. The mixture was poured into a 250 ml conical flask, containing autoclaved Phosphate buffered saline (0.01M; pH 7.2) and the bacterial concentration adjusted to 5x105 colony-forming units per milliliter (cfu/mL) using a spectrophotometer at a wavelength of 620nm. The suspension was further diluted to obtain a final concentration of 5x108 cfu/ml which is recommended for the study. Inoculation in the first screening was accomplished using the multiple needle method while inoculation in the second screening was done using the razor blade method (CIAT, 1987) (Plate 4). The experimental design employed was an alpha lattice which two replications, eight blocks and 10 lines/block.

Plate 4. Multiple needle and razor blade methods for CBB inoculation on common beans (CIAT-Uganda, 2012).

3.2.7. Data collection

The morphological data collected on these materials included number of pods, seed size (expressed as a weight in grams of 100 health counted seed per variety) and seed yield (expressed as a weight in grams of total harvested seed per variety). CBB reaction assessment scoring was done at 10, 35 and 56 days after the inoculation (CIAT, 1987). A scale of 1-9 as describedy van Schoonhoven and Pastor-Corrales (CIAT, 1987) was used to score the materials for CBB reaction; where: 1= is reserved for an absence of symptoms which is often equated with highest level of resistance and 9 = represents extreme susceptibility (presense of severe symptoms, damage, or stress). Finaly scores were used to group test lines into resistance categories. These categories were: Resistant (1-3), intermediate (4-6) and susceptible (7-9).

3.2.8. Data Analysis

Data were analysed using GenStat 14th Edition (John Nelder, Rothamsted Experimental Station, UK, 2011). All parameters measured including CBB severity scores, number of pots, seed yield and seed size of the 80 genotypes were subjected to Analysis of Variance (ANOVA) and means of significant treatments effects separated using LSD at 5% level of significance. ReML was used to save the entry means and get the Error in the alpha lattice design, with genotypes as a fixed effect and Rep/Block as random effects using the linear model (i):

$Yijk = \mu + Rj + Gi + B/Rjk + eijk.$ (i)

Where:

Yijk: total observation value (independent of Reps, Blocks and Genotypes effects)

- μ: Grand (Population) mean
- Rj: Replication effect
- Gi: Genotypes effects
- B/Rjk: Block per Reps effect
- Eijk: error effect

For seed yield and number of pods parameters where the alpha Lattice design analysis was not effective (Rep by block variances lower than the Lattice Effective Error), the data was re-analysed following a Complete Randomized Blocks Degign model (ii):

Yijk = μ + Rj + Gi + Bj+ eijk (ii)

Where:

- Yijk: total observation value (independent of Reps, Blocks and Genotypes effects)
- μ: Grand (Population) mean
- Rj: Replication effect
- Gi: Genotypes effects
- Bj: Block effect
- Eijk: error effect

In addition area under the disease progress curve (AUDPC) was calculated for each genotype according to Campbell and Madden (1990) as shown below:

$$AUDPC = \sum_{i}^{n-1}\left(\frac{y_i + y_{i+1}}{2}\right)(t_{i+1} - t_i)$$

Where, *Xi* is the severity rating of the host tissue damaged at the *i*th rating, *ti* is the time in days after inoculation at *i* the rating.

3.3. Results

3.3.1. Reaction of 80 bean lines to CBB

None of the screened genotypes was immune to infection by isolate "*Kawempe 1* " of *Xcp* although resistance to CBB varied among the screened lines (Table 2). At 10 DAI in both screening cycles, the analysis of variance (ANOVA) revealed no significant difference ($P<0.05$) among the 80 regionally grown common bean lines.

Table 2: ANOVA table showing Mean squares of CBB severity, AUDPC and relative AUDPC on 80 bean genotypes grown in Kawanda, Uganda in May-July and August-October, 2013.

First season of screening (May-July 2013)						
SOV	**d.f**	**CBB DAI 10**	**CBB DAI 35**	**CBB DAI 56**	**AUDPC**	**r-AUDPC**
Rep	1	0.100ns	5.256*	13.225*	7562.5*	0.044*
Rep/blk	14	0.587ns	0.581ns	1.426ns	1304.5ns	0.008ns
Gen	79	0.560ns	0.668*	0.961***	1703.0*	0.010*
Res	65	0.399ns	0.345ns	0.914**	720.2ns	0.004ns
LEE	65	0.421	0.371	0.973	781.2	0.005
Second season of screening (August-October 2013)						
Rep	1	0.756ns	0.306ns	5.63ns	716ns	0.004ns
Rep/blk	14	0.466ns	0.564ns	2.00ns	1259ns	0.007*
Gen	79	0.302ns	0.405ns	2.00ns	920ns	0.005ns
Res	65	0.266ns	0.438ns	1.43ns	658ns	0.004ns
LEE	65	0.288	0.455	1.50	719	0.004

Abreviations: SOV = Sources of variation, Rep. = Replications, d.f = Degree of freedom, Geno. = Genotypes, wt=weight, ws = weight of seed, AUDCP = Area under disease curve progress,r-AUDCP=Relative Area under disease curve progress, ns=not significant, *=Significant at 0.05, **=Significant at 0.01, Significant at 0.001.

Reaction to CBB means ranged between 1 to 8 on a 1-9 scale, with RUGANDURA being highest and JESCA lowest. CBB severity means of the lines at 35 and 56 DAI was significantly different only on the first cycle with P≤0.05 and P≤0.001, respectively (Table 2). RWV 1129 was highest and RWV 2070 lowest at 35 DAI.RW 846 was highest and RWV 2070 lowest at 56 DAI. AUDPC was also signifficant at (P≤0.05) only in the first cycle, being highest for RWV 3006 and lowest for RWV 1129 (Table 3).

Overall, CBB scores varied from 1 to 8 according to scale 1-9 used on this study, implying that the genotypes in the study were resistant to highly susceptible. In the first screening, 21 lines were considered resistanct to CBB (Table 3). When the screening was repeated, the reaction of the lines to CBB means scores also ranged from 1 to 8, implying that the genotypes were resistant to highly susceptible (Table 3). AUDCP was also not significantly affected by genotype (Table 2). It was highest on RWV 1129 and lowest on MCM 2001. During the second screening, the number of lines that were regarded resistant dropped in number to 10 (Table 3) .

Table 3. Reaction of 80 genotypes of common beans to CBB in first and second screening (May-July 2013)

First Screenig						**Second screening**				
Genotype	**CBB score 10 days**	**CBB score 35 days**	**CBB score 56 days**	**AUDPC**	**r-AUDPC**	**CBB score 10 days**	**CBB score 35 days**	**CBB score 56 days**	**AUDPC**	**r-AUDPC**
ACC 714	3.1	3.6	3.6	160.1	0.39	2.9	3.5	4.6	169.2	0.41
AFR 708	3.0	3.0	4.0	148.5	0.36	2.5	3.5	5.0	164.3	0.40
Agronome	3.0	4.5	5.1	195	0.47	3.1	3.6	5.0	175	0.42
CAB 2	**2.5**	**3.0**	**3.0**	**133.1**	**0.32**	2.6	4.0	5.0	176.1	0.43
CAL 143	3.0	3.0	3.5	143.3	0.35	3.0	3.5	4.5	165.3	0.40
CAL 96	3.1	3.4	5.4	172.7	0.42	3.5	3.5	5.5	183.4	0.44
CODMLB 001	4.0	4.5	6.1	218.8	0.53	3.5	4.0	6.5	202	0.49
CODMLB 003	**3.0**	**3.0**	**3.1**	**138.1**	**0.33**	3.1	4.0	6.1	195.7	0.47
DECELAYA 1	**3.0**	**3.0**	**3.1**	**138.4**	**0.40**	2.9	3.4	4.3	155.7	0.38
DOR 500	2.9	3.5	4.4	163.7	0.42	3.1	3.6	6.3	192	0.46
GARUKURARE	3.6	3.7	4.2	175.4	0.40	2.9	3.6	5.0	172.6	0.42
GASIRIDA	3.0	3.4	4.6	165.4	0.38	3.0	4.6	5.7	207.3	0.50
Gitanga 1	2.6	3.1	4.8	155.4	0.34	3.0	3.5	4.7	170.3	0.41

GLP 2	2.6	3.2	3.2	140.1	0.47	3.1	3.5	6.5	188.2	0.45
HM 21-7	3.4	4.0	6.0	196.4	0.39	2.5	3.5	5.0	164.1	0.40
ICYANA 2	3.0	3.5	4.0	160.1	0.50	2.9	3.5	5.0	171.5	0.41
JESCA	**1.0**	**2.1**	**3.0**	**138.1**	**0.33**	**1.9**	**2.6**	**3.0**	**135.9**	**0.32**
Kanyebwa	3.3	5.4	7.0	201	0.42	3.3	5.6	7.2	210.0	0.50
KAB06F2.8-12	3.0	3.5	3.6	155.1	0.37	2.6	3.5	4.6	162.2	0.39
KAB06F2.8-27	2.6	4.0	5.1	178.5	0.43	2.6	3.1	4.0	146.9	0.35
KAB06F2.8-36	4.1	4.0	5.6	203.1	0.49	4.0	4.1	6.5	213.6	0.52
KAB06F8.8-35	3.5	4.0	5.6	194.4	0.47	2.6	3.0	4.1	148.0	0.36
KAT 31	**1.5**	**2.0**	**3.0**	**131.7**	**0.32**	3.0	3.0	4.5	153.8	0.37
KAT 39	**2.0**	**2.6**	**3.0**	**125.5**	**0.30**	3.0	3.5	4.0	160.0	0.39
KAT 56	3.0	4.0	5.0	182	0.44	3.0	4.5	5.0	193.5	0.47
KAT 69	3.0	3.5	3.5	154.8	0.37	3.0	3.5	5.0	170.5	0.41
KIANGARA	4.2	4.2	5.8	210.8	0.51	3.1	4.1	5.2	191.8	0.46
KIVUZO	2.9	3.5	4.4	162.6	0.39	3.0	3.5	3.9	156.7	0.38
LMB 49	3.9	4.3	6.2	211.6	0.51	2.9	3.9	3.8	164.1	0.40
Local Yield Check	4.0	3.9	5.9	201.3	0.49	3.9	4.0	5.8	199.5	0.48
Local Yield Check high Fe	3.6	4.0	5.6	197.1	0.48	3.2	4.0	4.5	181.2	0.44
MAC 42	3.0	3.8	4.4	170.5	0.41	2.9	3.5	5.5	175.9	0.42
MAC 44	**3.0**	**3.0**	**3.4**	**140.8**	**0.34**	**2.8**	**2.9**	**3.3**	**136.5**	**0.33**
MAC 74	2.5	3.5	5.4	169.3	0.41	2.4	3.4	6.4	173.4	0.42

MAHARAJI SOJA	**2.0**	**3.0**	**3.3**	**131.4**	**0.32**	**1.4**	**2.9**	**3.4**	**191.1**	**0.46**
Massindi Yellow	3.0	4.5	7.0	170.5	0.41	2.5	4.0	6.0	186.3	0.45
MCB 49-89A	3.5	3.5	5.0	176.8	0.43	3.0	3.5	5.5	175.8	0.42
MCM 2001	**2.0**	**3.0**	**3.0**	**136**	**0.31**	**2.5**	**3.0**	**3.0**	**131.8**	**0.32**
MIB 456	**1.0**	**2.0**	**3.0**	**138.1**	**0.33**	**1.5**	**2.0**	**3.0**	**137.8**	**0.33**
MONTACALM	**3.1**	**3.0**	**3.1**	**141.4**	**0.34**	2.5	3.1	4.5	150.9	0.36
NABE 3	2.4	2.8	3.8	133.3	0.32	2.5	3.0	5.5	156.9	0.38
Nain De Kyondo	2.5	3.0	3.6	137.1	0.33	2.9	4.5	5.5	196.7	0.48
NDIMIRAKUGUJA VOL	3.5	4.1	5.6	197.5	0.48	3.4	4.0	6.0	199.8	0.48
Ngwaku-Ngwaku	3.6	3.4	5.9	184.2	0.44	3.5	4.0	6.0	200.2	0.48
NGWINxCAB2/2/3/1/1	3.0	3.1	3.5	146.1	0.35	2.5	3.4	4.0	148.2	0.36
NUA 45	**2.1**	**2.6**	**3.0**	**170.6**	**0.41**	**1.4**	**2.2**	**3.0**	**160.9**	**0.37**
NUA 59	3.5	4.0	4.5	183.9	0.44	2.5	3.5	5.5	169.1	0.41
NUA 69	3.0	4.4	5.9	198.3	0.48	2.9	3.5	4.5	161.9	0.39
NUA 99	3.6	4.0	5.6	196.8	0.48	3.5	4.0	4.9	187.5	0.45
NUV 219-1	2.6	3.5	5.0	164.9	0.40	2.9	3.5	4.5	163.8	0.40
ROBA 1	1.9	3.0	3.9	132.8	0.32	3.1	3.5	4.5	167.4	0.40
RUGANDURA	4.5	4.9	6.9	231.7	0.56	3.5	4.5	6.4	216.3	0.52
RW 1180	2.5	3.5	4.1	156.1	0.38	3.0	4.5	6.5	207.3	0.50
RW 184	2.9	3.3	3.7	149.9	0.36	2.4	3.4	4.8	156.8	0.38

RW 547	**2.9**	**3.0**	**3.0**	**135.7**	**0.33**	2.6	3.0	4.5	149.4	0.36
RW 582	3.5	4.1	5.0	191.1	0.46	3.5	3.9	6.0	193.2	0.47
RW 805	2.9	3.4	4.9	165.6	0.40	3.6	3.5	4.6	174	0.42
RW 846	3.4	5.4	8.0	216.5	0.52	3.6	5.5	7.4	171.6	0.41
RWR 719	3.0	3.0	3.5	143.3	0.35	3.0	3.5	4.0	160	0.39
RWV 1129	4.0	5.4	6.4	242.2	0.59	4.0	4.5	6.4	222.6	0.54
RWV 2070	**1.1**	**2.0**	**2.6**	**132.1**	**0.32**	**1.9**	**2.8**	**3.0**	**137.7**	**0.32**
RWR 2154	**1.0**	**2.6**	**3.1**	**165.1**	**0.40**	**2.0**	**2.6**	**3.2**	**172.6**	**0.42**
RWV 2245	3.0	3.1	3.1	141.6	0.34	2.6	3.5	5.0	164.7	**0.40**
RWV 2359	3.6	4.1	6.1	204.3	0.49	3.6	4.0	6.0	198.1	0.48
RWV 2361	3.1	4.0	5.6	190.5	0.46	3.0	3.5	6.4	185.5	0.45
RWV 2887	3.1	4.7	5.7	207.9	0.50	3.1	3.6	5.3	181.5	0.44
RWV 3006	**2.0**	**2.4**	**2.9**	**111.6**	**0.27**	**1.9**	**2.5**	**3.4**	**167.9**	**0.41**
RWV 3316	3.4	3.8	5.7	189.9	0.46	2.9	3.5	4.9	166	0.40
SMC 16	**2.9**	**3.0**	**2.9**	**134.8**	**0.33**	3.1	4.0	4.5	178.9	0.43
SMC 17	**3.0**	**3.0**	**3.5**	**142.8**	**0.35**	3.0	3.5	4.4	162	0.39
SMC 18	3.0	3.6	5.1	174.1	0.42	3.1	4.0	5.5	187.7	0.45
SMC 21	**2.9**	**3.0**	**2.9**	**134.2**	**0.32**	2.9	3.4	5.3	166.1	0.40
USDK-CBB-15	**2.0**	**3.0**	**2.9**	**128.3**	**0.31**	**2.0**	**2.8**	**3.4**	**142.1**	**0.34**
VAX 1	3.5	4.0	5.4	190.5	0.46	3.5	4.0	6.5	202.7	0.49
VAX 2	**2.0**	**3.0**	**3.4**	**140.3**	**0.34**	1.5	3.5	4.0	152.5	0.37
VAX 3	3.4	4.0	5.9	195.3	0.47	3.0	4.0	5.9	188.1	0.45

VAX 4	3.5	3.5	5.1	176.3	0.43	2.5	3.5	6.1	178.1	0.43
VAX 5	3.9	4.0	5.9	202.8	0.49	3.0	4.0	6.5	196.3	0.47
VCB 81013	3.0	3.5	4.9	168	0.41	2.4	2.9	6.9	167	0.40
VRA 4	3.0	3.9	4.4	171	0.41	2.4	3.0	3.5	133.6	0.32
Zebra 4	2.0	2.5	4.1	126	0.30	3.1	5.1	6.0	220	0.53
Means	**3.1**	**3.6**	**4.5**	**167.6**	**0.4**	**2.9**	**3.6**	**5.1**	**173.7**	**0.4**
LSD	**1.3**	**1.2**	**1.2**	**56.2**	**0.1**	**1.1**	**1.4**	**2.5**	**53.9**	**0.1**
CV %	**20.9**	**16.9**	**13.5**	**16.7**	**17.7**	**18.6**	**18.7**	**24.0**	**15.4**	**15.1**

Abreviations: **LSD**= Least significant Difference at 5 %, **CV**= Coefficient of variation in percentage

3.4. Reponse of the ACC lines to common bacterial blight (Isolate Kawempe 1) in Uganda

Analysis of variance revealed that genotypes were significantly different ($P \leq 0.05$) at all the scoring dates apart from 10 days after inoculation (Table 4). Similar results were obtained for the 80 regional nutritional nursery genotypes. Disease severity was highest in ACC 1, ACC 15 and VAX1 at 10 DAI, in ACC 7 and ACC 11 at 14 DAI, in Masindi Yellow and Kanyebwa at 19 DAI and lowest in ACC 12, ACC 14, ACC 18 at 10 DAI, in ACC 22 at 19 DAI, ACC 6, ACC 10 at 19 DAI.Was also significant different the AUDPC and r-AUDPC at ($P \leq 0.01$).

Table 4: Analysis of varience and means squares and probability for the 24 ACC , including six VAX lines and three local check lines and genotypes.

SOV	Df	10 DAI	14 DAI	35 DAI	AUDPC	r-AUDPC
Rep	1	0.0606	1.8333	0.242	286.5	0.00980
Genotype	32	0.3277ns	1.4896*	5.555*	781.2**	0.02672**
Residual	32	0.2481	0.8021	2.117	297.7	0.01018
Total	65					

Abreviations: **SOV**=Source of variance, **df**=Degree of Freedom, **DAI**=Days After Inoculation, **CV %**= Coefficient of Variation in percentage, *****=Significant at 0.05, ******=Significant at 0.01, *******=Significant at 0.001, **ns**= Not Significant

CBB scores ranged from 1 to 8, meaning that the response to CBB was between resistant to highly susceptible according to the 1-9 scale used. The lines ACC 10, ACC 16, ACC 18, ACC 21, ACC 22, ACC 3, ACC 4, ACC 5 and MCM 2001 had the least CBB severity at 35 days. r-AUDPC ranged from 0.26 - 0,64 (Table 5). Genotypes that recorded the highest diseases were ACC 11, Kanyebwa, ACC 7, VAX 4 and Masindi Yellow, all of which had CBB score at 35 days variying from 7 to 8. For these genotyepes, the r-AUDPC values ranged from 0,57 - 0.64.

Table 5: Means of CBB affects on the ACC lines

Lines	10 DAI	14 DAI	35 DAI	AUDPC	r-AUDPC
ACC 1	2.5	2.5	3.5	54.50	0.32
ACC 10	**2.0**	**2.5**	**2.5**	**46.20**	**0.27**
ACC 11	2.0	5.0	7.0	101.50	0.59
ACC 12	1.0	4.0	5.0	75.50	0.44
ACC 13	2.0	4.5	6.5	93.20	0.55
ACC 14	1.0	2.5	4.0	54.20	0.32
ACC 15	2.5	3.0	4.5	66.20	0.39
ACC 16	**1.5**	**3.0**	**3.0**	**56.80**	**0.33**
ACC 17	1.5	2.5	3.5	48.50	0.28
ACC 18	**1.0**	**2.5**	**2.5**	**43.80**	**0.26**
ACC 19	2.0	3.0	4.5	65.00	0.38
ACC 2	2.0	3.5	4.5	69.80	0.41
ACC 20	2.0	3.5	5.5	76.80	0.45
ACC 21	**1.5**	**2.5**	**3.0**	**48.50**	**0.28**
ACC 22	**2.0**	**2.0**	**3.0**	**45.00**	**0.26**
ACC 23	2.0	3.0	5.5	72.00	0.42
ACC 24	2.0	2.5	3.5	53.20	0.31
ACC 3	**2.0**	**2.5**	**3.0**	**49.80**	**0.29**
ACC 4	**1.2**	**2.5**	**3.0**	**48.50**	**0.28**
ACC 5	**2.0**	**2.5**	**3.0**	**49.80**	**0.29**
ACC 6	2.0	2.5	4.5	60.20	0.35
ACC 7	2.5	5.0	7.0	102.80	0.60
ACC 8	1.5	2.5	3.5	52.00	0.30
ACC 9	1.5	3.5	6.0	79.00	0.46

Kanyebwa	2.0	4.5	7.5	100.20	0.57
M Yellow	2.0	5.0	8.0	108.50	0.64
MCM 2001	**2.0**	**2.5**	**3.0**	**49.80**	**0.29**
VAX 1	2.5	3.5	6.0	81.50	0.48
VAX 2	2.0	3.5	6.0	80.20	0.47
VAX 3	1.5	2.5	4.0	55.50	0.33
VAx 4	2.0	4.0	7.0	92.00	0.54
VAX 5	2.0	3.5	6.5	83.80	0.49
VAX 6	1.5	2.5	2.5	45.00	0.26
Means	**1.85**	**3.17**	**4.61**	**66.90**	**0.39**
LSD	**1.02**	**1.82**	**2.96**	**35.15**	**0.21**
CV %	**26.90**	**28.30**	**31.60**	**25.80**	**25.80**

Abreviations: **LSD**= Least significant Difference at 5 %, **CV**= Coefficient of variation in percentage

3.5. Reponse of the selected lines to six different isolates for CBB and assessment of virulence on the isolate Kawempe 1).

The analysis of variance revealed that the isolates were significantly different in all scoring dates except at 35 DAI, the analysis of analyze of variance also revealed that the Genotypes were significanttly different in all scoring dates with strong significance at 35 and 56 DAI ($P<0.001$). The results also revealed that interaction between isolate and genotype were not significant ($P>0.05$) in none of the scoring dates (Table 6).

Table 6. Analysis of variance and Means square of the isolates, Genotypes and Isolate x Genotypes across all different scoring dates

SOV	**d.f**	**10 DAI**	**14 DAI**	**35 DAI**	**56 DAI**	**AUDPC**	**r-AUDPC**
Replication	1	0.2	0.22	0.17	5.16	50.30	0.000
Isolate	5	0.68*	1.51**	2.23	4.55*	5462.20***	0.032**
Genotype	10	0.52**	0.45*	2.61**	28.58*	5941.40*	0.034***

					*	**	**	
Isolate	x	50	0.13ns	0.18ns	0.42ns	0.61ns	811.00	0.004
Genotype								
Residual		58	0.17ns	0.18ns	0.28ns	0.54ns	843.70	0.004
Total		**129**						

Abreviations: **SOV**=Source of variance, **df**=Degree of Freedom, **DAI**=Days After Inoculation, **CV %**= Coefficient of Variation in percentage, *****=Significant at 0.05, ******=Significant at 0.01, *******=Significant at 0.001, **ns**= Not Significant

The "*Kawenpe 1*" isolate was the most aggressive across all the scoring dates, which the scoring ranging from 1.56 to 5.00 and the isolate CBB1 showed the same pattern up to 35 DAI (Table 7)

Table 7. Means for the isolates across the scoring dates

Isolate	**10 DAI**	**14 DAI**	**35 DAI**	**56 DAI**	**AUDPC**	**r-AUDPC**
CBB 1	1.41	1.59	2.86	3.96	124.40	0.30
Kawenpe 1	1.56	1.86	3.27	5.00	147.60	0.36
KIS-wa-001	1.18	1.27	2.46	3.73	109.00	0.26
Kyanga	1.23	1.32	2.41	3.96	111.00	0.27
MA-F-011	1.14	1.14	2.6	4.02	105.10	0.25
MSD-B-05	1.09	1.33	2.71	4.35	112.80	0.27
Grand mean	**1.27**	**1.42**	**2.72**	**4.19**	**118.30**	**0.29**
LSD	**0.29**	**0.28**	**0.67**	**0.66**	**13.75**	**0.03**
CV%	**32.2**	**30.2**	**19.5**	**17.5**	**24.60**	**24.60**

Abreviations: **DAI**=Days After Inoculation, **LSD**= Last Significant Difference

The results also shows that the susceptible checks (Kanyebwa and Masindi yellow) used in this experiment had the higest score in all the scoring dates with the scores varying from 1.5 to 7.3 for Kanyebwa on the 10 to 56 DAI respectively, for Masindi yellow the scores varied from 2.0 to 7.72 at 10 and DAI 56 respectively (Table 8).

Table 8. Means for the genotypes and lines across the scoring dates

Genotype	10 DAI	14 DAI	35 DAI	56 DAI	AUDPC	r-AUDPC
JESCA	1.2	1.3	2.6	3.1	105.60	0.25
Kanyebwa	1.5	1.6	3.4	7.3	170.70	0.41
Masindi yellow	2	1.8	3.7	7.2	148.60	0.36
MCM 2001	1.2	1.3	2.6	3.3	108.20	0.26
MIB 456	1.3	1.3	2.5	3.3	105.60	0.26
NUA 45	1	1.3	2.4	3.3	102.50	0.25
RWR 2070	1.2	1.5	2.4	3.3	106.00	0.26
RWR 2154	1.1	1.1	2.5	3.4	104.10	0.25
VAX 1	1.3	1.5	2.3	3.5	105.40	0.25
VAX 2	1.3	1.5	2.5	4.1	116.60	0.28
VAX 3	1.3	1.5	3	4.2	128.00	0.31
Grand mean	**1.2**	**1.4**	**2.7**	**4.1**	**118.30**	**0.29**
LSD	**0.33**	**0.35**	**0.43**	**0.6**	**23.72**	**0.03**
CV%	**32.20**	**30.20**	**19.50**	**17.50**	**24.60**	**24.60**

Abreviations: **DAI**=Days After Inoculation, **LSD**= Last Significant Difference

The results also revealed that the resistant checks (VAX 1, VAX 2 and VAX 3) in general were moderately resistant with the scores varying from 3.5, 4.1 and 4.2 respectively. The lines **JESCA, RWV 2070, RWR 2154, MIB 456, NUA 45** and **MCM 2001** appeared to be more resistant to the disease given that they had scores ranging from 3.1 to 3.4 suggesting that there were resistant to moderate resistant according to scale.

In general on the first scoring date 10 DAI the genotypes did not show the symptoms for almost all the isolates according to the, except for Kawempe 1 and CBB 1. Almost the same pattern was observed on second score at 14 DAI, all the genotypes showed the symptoms of CBB (Table 9). The results also revealed that at the 56 DAI, the susceptible check (Kanyebwa and Masindi

yellow) had high score for all the isolates, ranging from 6.5 to 8.5 meaning that they shown to be susceptible. The resistant checks (VAX 1, VAX 2 and VAX 3) appeared to be moderately resistant for the isolate Kawempe 1. From this experiment, the results revealed that the lines JESCA, MIB 456, NUA 45 and RWR 2070 appeared to be resistant for at least 4 of the 6 isolates used on this experiment with the maximum score of 3 and the most virulent isolate was Kawempe 1.

Table 9. Means of the genotypes for each isolate across the scoring date

Genotypes											
10 DAI											
Isolates	**JESCA**	**Kanyebwa**	**Massindyellow**	**MCM 2001**	**MIB 456**	**NUA 45**	**RWR 2070**	**RWR 2154**	**VAX 1**	**VAX 2**	**VAX 3**
CBB1	1.0	1.5	1.5	1.5	1.5	1.0	1.5	1.0	1.5	2	1.5
kawenpe 1	1.5	2.0	2.0	1.5	2.0	1.0	1.0	1.5	1.5	1.5	1.5
KIS-wa-001	1.0	1.5	2.0	1.0	1.0	1.0	1.0	1.0	1.5	1.0	1.0
Kyanga	1.5	1.5	2.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
MA-F-011	1.0	1.5	1.1	1.0	1.0	1.0	1.0	1.0	1.5	1.0	1.5
MSD-B-05	1.0	1.0	2.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
14 DAI											
CBB1	1.0	1.5	1.5	1.5	1.5	1.0	1.5	1.0	1.5	2.0	1.5
kawenpe 1	1.5	2.0	2.0	1.5	2.0	1.0	1.0	1.5	1.5	1.5	1.5
KIS-wa-001	1.0	1.5	2.0	1.0	1.0	1.0	1.0	1.0	1.5	1.0	1.0
Kyanga	1.5	1.5	2.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
MA-F-011	1.0	1.5	1.1	1.0	1.0	1.0	1.0	1.0	1.5	1.0	1.5
MSD-B-05	1.0	1.0	2.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
35 DAI											
CBB1	4.0	4.0	4.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	3.0
kawenpe 1	3.0	4.5	4.5	3.0	3.0	3.0	2.5	2.0	3.0	3.0	4.5
KIS-wa-001	3.0	2.5	3.0	3.0	2.5	3.0	2.0	2.0	2.0	2.5	2.5
Kyanga	2.5	3.0	3.0	2.0	2.0	2.0	2.5	2.5	2.0	2.5	2.0
MA-F-011	2.0	3.5	4.1	2.0	2.5	2.5	2.0	2.5	2.0	2.0	3.5

MSD-B-05	2.5	3.0	3.4	3.0	2.5	2.0	3.0	3.5	2.0	2.5	2.5
56 DAI											
CBB1	3.0	7.0	7.0	3.5	3,0	3.0	3.0	3.0	3.0	4.0	4.0
kawenpe 1	3.0	8.5	8.5	4.0	4.0	4.0	3.0	4.0	5.0	5.0	6.0
KIS-wa-001	3.0	7.5	6.5	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0
Kyanga	3.0	7.5	6.5	3.0	3.0	3.0	3.0	3.5	3.5	4.0	3.5
MA-F-011	3.0	6.5	7.2	3.0	3.0	3.0	3.0	3.0	3.5	3.5	5.5
MSD-B-05	3.5	6.5	7.4	3.5	4.0	3.5	4.5	4.0	3.0	5.0	3.0

Abreviations: **DAI**=Days After Inoculation

3.6. Effect of CBB infection on yield performance under screen house conditions

The analysis of variance on the first screening revealed that the genotypes were significantly different for all the yield parameters under analysis at (P≤0.001). However, for the second screening, the effect of genotypes was only significant for 100 seed weight at (P≤0.001) (Table 10).

Table 10 Analise of variance the yield parameters on first and second screening.

First season of screening (May-July 2013)				
SOV	**d.f**	**Seed yield (gm)**	**N of pods**	**100 seed weight (Seed size) (gm)**
Rep	1	14.050	6.320	0.23
Rep/blk	14	-	-	-
Gen	79	98.860***	33.750***	259.66***
Res	65	30.810	12.690	0.09
LEE	65			
Second season of screening (August-October 2013)				
Rep	1	1373.07	343.84**	0.23
Rep/blk	14		26.81ns	
Gen	79	60.04ns	21.34ns	259.66***
Res	65	50.84	21.21ns	0.08
LEE	565		22.47	

Abreviations: Rep=Replication; Gen=Genotypes; Blk=Blocks; wt=weight, ws = weight of seed, ns=not significant, *=Significant at 0.05, **=Significant at 0.01, Significant at 0.001.

On the first screening the seed yield ranged from 10 to 65.5 grams. It was highest in GASIRIDA followed by RUGANDURA, VCB 81013 and Zebra 4. These weighed 65.50, 35.04, 30.94 and 30.86 gm respectively.

The lowest weight was recorded in AFR 708 followed by KAT 31, RWV 2887 and Ngwaku-Ngwaku, with weight of 10.00, 10.10, 10.27 and 10.44 gm respectively. Pod number per plant ranged from 1 to 20.33 pods, with ACC 714 being highest followed by RUGANDURA, VCB 81013 and Zebra 4. These had 20.33, 20.30, 19.42 and 19.36 pods respectively. The lowest number of pods were recorded on KAT 69 followed by KAB06F8.8-35, RWR 3316 and Masindi Yellow with values of 1, 5.38, 5.52 and 6 pods respectively. The 100 seed weight ranged from 20 to 59 grams, with Local yield check being highest while the lowest was recorded on KAT 69 (Table 11).

On the second screening, seed yield ranged from 4.62 to 61.81 grams, with MAHARADJI SOJA being highest with a weight of 61.81 gm. It was lowest in CAL 96 follwed by VAX 5, CODMLB 003 and NUA 99 with values of 4.62, 6.20, 6.94 and 10.01 gm respectively. Pod number also varied among genotypes. It was lowest in GITANGA 1 which had an average of 3.57 pods and highest in MCB 49-89A with mean pod number of 29.50. The 100 seed weight ranged from 19 to 58.41 grams, being highest in Loca check yield followed by KIANGARA, Zebra 4 and MAC 74, weighing 58.41, 58.00 and 56.65 gm respectively and lowest in KAT 69 with a weight of 19.00 gms (Table 11).

Tabel 11: **Means for the yield parameters on the first and second screening**

First Screening				**Second Screening**		
Genotype	**Seed yield (gm)**	**N of pods**	**100 seed weight (Seed size) (gm)**	**Seed yield (gm)**	**N of pods**	**100 seed weight (Seed size) (gm)**
ACC 714	23.9	20.33	26.04	16.49	12.88	25.04
AFR 708	10.00	10.25	34.48	11.70	12.50	33.48
Agronome	18.75	14.36	31.92	21.20	10.94	30.92
CAB 2	12.50	8.63	36.47	13.34	8.81	35.47

CAL 143	12.85	11.00	37.00	22.10	10.50	36.00
CAL 96	17.10	7.43	44.29	4.62	5.82	43.29
CODMLB 001	12.84	10.08	37.81	15.34	10.89	36.81
CODMLB 003	23.10	12.65	46.76	6.94	9.14	45.76
DECELAYA 1	12.45	12.34	36.50	16.16	7.60	35.50
DOR 500	29.95	18.71	21.85	10.40	8.62	20.85
GARUKURARE	20.75	18.54	32.55	23.48	12.44	31.55
GASIRIDA	65.50	18.38	44.33	13.13	8.62	43.33
Gitanga 1	23.50	11.56	24.79	16.87	3.57	23.79
GLP 2	17.15	15.67	42.38	15.43	5.34	41.38
HM 21-7	15.40	13.75	43.64	16.19	13.11	42.64
ICYANA 2	12.15	8.76	39.65	10.95	6.83	38.65
JESCA	27.50	19.19	32.37	13.82	13.31	31.37
KAB06F2.8-12	16.50	7.34	50.63	19.19	10.64	49.63
KAB06F2.8-27	16.20	13.34	42.82	25.00	15.54	41.82
KAB06F2.8-36	18.20	8.51	38.42	16.08	9.32	37.42
KAB06F8.8-35	18.65	5.38	31.11	24.72	17.15	30.11
KAT 31	10.10	8.50	57.20	23.75	12.50	56.20
KAT 39	12.05	12.50	53.48	21.75	15.5	52.48
KAT 56	12.35	8.50	30.00	18.25	12.50	29.00
KAT 69	24.0	1.00	20.00	19.85	12.50	19.00
KIANGARA	24.12	12.74	59.89	24.98	14.00	58.00
KIVUZO	16.49	14.07	35.45	18.51	7.02	34.45
LMB 49	27.57	18.88	36.22	14.84	12.65	35.22
Local Yield Check	10.88	7.01	59.41	13.40	6.15	58.41
Local Yield Check high Fe	18.45	7.52	53.64	18.38	7.27	52.64
MAC 42	12.46	8.75	44.78	19.17	11.94	43.78
MAC 44	22.19	12.42	54.71	17.08	14.22	53.71
MAC 74	17.36	8.35	57.50	11.20	11.15	56.50
MAHARAJI SOJA	16.06	15.76	22.86	61.81	15.77	21.86

Masindi yellow	15.65	6.00	32.00	11.30	8.00	31.00
MCB 49-89A	23.35	16.00	40.81	53.85	29.50	39.81
MCM 2001	16.55	14.00	25.61	16.35	11.50	24.61
MIB 456	16.18	14.26	24.63	43.64	23.61	23.63
MONTACALM	13.15	10.06	50.43	21.28	16.32	49.43
NABE 3	20.48	15.17	25.38	13.16	9.64	24.38
Nain De Kyondo	24.02	14.65	32.17	12.62	8.81	31.17
NDIMIRAKUGUJA VOL	25.14	15.69	37.64	19.05	11.83	36.64
Ngwaku-Ngwaku	10.44	9.93	46.43	11.22	7.32	45.43
NGWINxCAB2/2/3/ 1/1	15.67	12.19	23.33	14.43	12.34	22.33
NUA 45	13.65	10.33	48.26	19.29	15.88	47.26
NUA 59	19.70	11.45	27.57	14.58	11.09	26.57
NUA 69	19.99	13.86	44.44	15.71	14.39	43.44
NUA 99	12.61	12.33	45.59	10.01	10.20	44.59
NUV 219-1	21.20	14.99	33.75	12.52	12.89	32.75
ROBA 1	11.02	7.57	23.90	15.94	11.85	22.90
RUGANDURA	35.04	20.30	29.80	11.92	10.09	28.80
RW 1180	20.99	13.08	30.45	18.98	13.35	29.45
RW 184	16.62	15.88	30.94	16.34	13.65	29.94
RW 547	16.79	13.75	33.77	11.28	10.84	32.77
RW 582	19.22	11.19	40.34	14.48	10.34	39.34
RW 805	24.45	14.73	26.92	21.89	15.44	25.92
RW 846	18.21	16.43	27.20	11.07	10.36	26.20
RWR 719	18.00	12.50	26.21	24.05	13.50	25.21
RWV 1129	20.94	12.80	45.45	17.57	10.64	44.45
RWV 2070	10.81	14.82	21.11	18.91	11.64	20.11
RWR 2154	19.11	7.48	53.33	12.07	9.32	52.33
RWV 2245	12.33	15.56	40.00	15.76	11.39	39.00
RWV 2359	14.42	11.27	40.61	16.39	12.77	39.61

RWV 2361	16.61	11.83	37.44	14.56	9.20	36.44
RWV 2887	10.27	8.54	36.51	17.45	9.62	35.51
RWV 3006	17.19	11.15	31.84	17.69	10.20	30.84
RWV 3316	14.28	5.52	22.50	14.18	9.70	21.50
SMC 16	10.12	8.07	36.36	19.29	8.85	35.36
SMC 17	14.06	12.00	29.36	17.98	7.09	28.36
SMC 18	12.68	10.56	30.37	13.56	9.89	29.37
SMC 21	13.18	12.28	27.69	17.59	9.52	26.69
USDK-CBB-15	19.73	7.97	44.44	20.76	15.85	43.44
VAX 1	17.47	12.43	23.93	14.96	15.41	22.93
VAX 2	22.72	15.93	29.64	18.76	7.91	28.64
VAX 3	21.73	16.97	30.92	11.66	17.85	29.92
VAX 4	19.00	13.16	26.76	14.90	13.15	25.76
VAX 5	13.94	14.11	23.27	6.20	6.40	22.27
VCB 81013	30.94	19.42	43.33	19.90	13.87	42.33
VRA 4	20.64	13.86	31.51	12.76	9.39	30.51
Zebra 4	30.86	19.36	57.65	15.55	11.94	56.65
Means	**16.40**	**12.40**	**37.10**	**14.00**	**10.70**	**37.10**
LSD	**11.10**	**7.10**	**0.60**	**14.20**	**9.50**	**0.60**
CV %	**34.00**	**28.70**	**0.90**	**51.00**	**43.80**	**0.90**

Abreviations: **LSD**= Least significant Difference at 5 %, **CV**= Coefficient of variation in percentage

3.5. Discussion

Identification of sources of resistance to CBB has been a major objective of many researchers involved in common bean breeding programs around the world. Although there are no bean line that are immune to CBB (Sherf and MacNab, 1986), they have varying levels of resistance and a reasonable levels have been identified in some genotypes implaying that ther is potential of improving CBB resistance in beans.

In this study, several bean genotypes from different populations were evaluated for their reaction to CBB. Results of this study revealed that at 10 DAI beans varieties were not significantly

different in terms of CBB severity. An important finding in this study is that some of the lines screened in this study had higher CBB resistance levels in comparison to some of the lines known to be resistant; which in some cases have even been used as source of resistance (Singh and Muñoz, 1999; Zapata *et al.*, 1998; Jara *et al*., 1999). It is known that East Africa has both *Xanthomonas campestris* pv. *phaseoli* and *Xanthomonas campestris* pv. *phaseoli* var. *fuscans.* The isolate *"Kawempe 1"* used in this study is a *X. campestris* pv. *phaseoli* var. *fuscans.* There are a number of reports indicating that the *X. campestris* pv. *phaseoli* var. *fuscans* isolates tend to be more pathogenic to beans (Opio *et al*., 1996). Therefore, the choice of a "*fuscans*" strain in this study was based on these reports.

The set of genotypes studied, were screened in two cycles. On the first screening and at 56 DAI 21 genotypes were considered resistant, However, for the second screening, only 10 genotypes were considered resistant. It is worth to note that the first screening was accomplished using a "multiple needle" method, while in the second screening inoculation was carried out using "razor blade" technique. Silva *et al*. (2009), reported that "multiple needle" was a better menthod compared with to the "razor blade" method. There are reports suggesting that environmental conditions exert influence on genotypes response to *Xcp* inoculation (Ferreira *et al.,* 2003), implying that in this study regardless of the method used for inoculation the specific environmental conditions in each cycle might have played important role on the genotypes reaction to *Xcp* infestaction. For instance, also the genetic backgroung of the genotypes in terms of growth habit has its influence on the there response to *Xcp* infestaction. Climbing bean with its vigorous vegetative growth, which often is clinging on to stakes, spreading its canopy in the aerial space, while bush with their canopy crowded close to the ground level, and experiencing a different microclimate (Chataika *et al.,* 2011). However, the results of this study are similar with those reported from other autors, where differences on the cultivars responses where found (Maringoni *et al.,* 1993; Rava & Sartorato, 1994; Torres and Maringoni, 1999). Is important to mention that the genotypes RWV 2070, RWR 2154, MCM 2001, NUA 45, JESCA, MIB 456, MAHARAJI SOJA, RWV 3006, USDK-CBB-15 and MAC 44 had almost the same rating 56 DAI for resistance in two cycles of screening.

3.6. Sectional conclusion and recomendation

In general, the results of this study showed the importance of knowing the reaction of genotypes, since the reaction of many of the materials used in this study was not yet known. In addition, the inoculation methods used were efficient for determining resistance and susceptibility reaction of the beans genotypes to the *Xcp "Kawenpe 1"* isolate. The method of multiple needles proved to be the most practical during inoculation. Also from the results of this study can be concluded that symptom development in these cultivars depended on the methods of inoculation, specific environmental conditions on each one of the two cycles and bean genotype growth habit.

In this study, most genotypes were categorized as susceptible, where only 21 out 80 were resistant in the first cycle and on the second cycle only 10 were resistant. The genotypes RWV 2070, RWR 2154, MCM 2001, NUA 45, JESCA and MIB 456, appeared to be more consistant on their response to the CBB inoculation, they had almost the same rating at 56 DAI in both screening cycles, eventough using a different inoculation methods in each screening. The significance of this study is in presenting the potential sources of tolerance to CBB, which can be used in plant breeding programs to introgress genes conferring resistance into a local adapted and farmer preferred genotypes.

CHAPTER FOUR

GENETIC MECHANISMS OF RESISTANCE TO COMMON BACTERIAL BLIGHT AMONG AFRICAN REGIONAL BEAN GERMPLASM

4.1. Introduction

Determination of the mode of inheritance is useful in the development of plant cultivars with improved traits, such as resistance to biotic and abiotic stresses. In common bean breeding for resistance to biotic and abiotic stresses, knowledge of the mode of inheritance allows the breeder to determine whether the incorporated resistance is durable and can be expressed in different environments (Silva *et al.,* 2009). By estimating heritability of a given trait, the fraction of total phenotypic variation explained by additive genetic effects may be determined. This information is important especially in improving self-pollinated crops such as beans as it allows breeders to adopt appropriate selection strategies and to predict rates of phenotypic change (Falconer and Mackay, 1996). In this study, inheritance of common bacterial blight disease (CBB) resistance in newly identified resistant genotypes (discussed in chapter III of this thesis) was investigated. Genetic parameters such as the general combining ability (GCA) and specific combining ability (SCA) were determined to help explain the mechanisms of CBB resistance in these genotypes.

4.2. Materials and methods

4.2.1. Experimental design and crosses used in the study

A total of 24 single crosses were made at the International Centre for Tropical Agriculture (CIAT) Uganda station based at the National Agricutural Research Laboratories (NARL) at Kawanda.

The parents for the crosses involved six resistant varieties identified under Study 1: MIB 456, MCM 2001, RWR 2154, RWV 2001, JESCA and NUA 45. These were crossed with four locally adapted but CBB susceptible varieties; Kanyebwa, Masindi Yellow, K131 and K 132 (CAL 96).

All the parents are currently regionally grown as released varieties and/or breeding parents in Eastern, Central and Southern Africa.

The mating design used in this study was North Carolina II with the *resistant* lines being the male parents and *susceptible* varieties as female parents.The study was conducted under screen house conditions. The parents for each line were planted in individual pots in sterile soil. Five pots were used per parents with five seeds per pot. Four crossing blocks were established in a time staggered way to ensure synchronisation of flowering dates of the parents. From the 24 expected crosses only 12 were successful, they included K 131 x JESCA, K 131 x MCM 2001, K 131 x MIB 456, K 131 x RWV 2070, K 132 x JESCA, K 132 x MIB 456, K 132 x RWV 2070, KANYEBWA x JESCA, KANYEBWA x RWV 2070, Masindi Yellow X RWV 2070, Masindi yellow x MCM 2001 and Masindi yellow x MIB 456. Seed was harvested and advanced to F2 generation. The F2 seed was planted in two replications, with three pots per replication and five seed per pot, making 30 seed per population. One week after emergence, F2 plants were tagged and numberd in each replication for easy identification during disease assessments. The parental lines used in this study are described in Table 12 below:

Table 12. Description of parental lines

Entry	CBB at 56 DAI (1-9)	Seed colour	Growth habit	Genepool	Souce
MIB 456	3	Black	Climber	Midlle America	PABRA
MCM 2001	3	Red	Climber	Midlle America	PABRA
NUA 45	2	Red and Cream	Bush	Andrean	PABRA
JESCA	3	White	Climber	Midlle America	PABRA
RWR 2154	3	Cream and Pink	Bush	Andrean	PABRA
RWV 2070	2	Gray	Climber	Andrean	PABRA
Masindi	7	Yellow	Bush	Andrean	PABRA

Yellow					
Kanyebwa	7	Pink and Red	Bush	Andrean	PABRA
K131	6	Cream and gray	Bush	Midlle America	PABRA
K132	6	Cream and gray	Bush	Andrean	PABRA

PABRA= Pan-African Beans Research Alliance

4.2.2. Data collection and Analysis

Data were collected on morphological traits and disease accurrence. The morphological data included; number of pods per plant, seed size, weight of total seed harvested per cross expressed. CBB severity was scored at 10, 35 and 56 days after the inoculation (CIAT, 1987). The inoculums production and the inoculation was done as described on the chapter III if this thesis. Data were analysed using GenStat 14th Edition (John Nelder, Rothamsted Experimental Station, UK, 2011).
A scale of 1-9 as described by van Schoonhoven and Pastor-Corrales (CIAT, 1987) was used to score CBB severity; where 1= is reserved for an absence of symptoms which is often equated with highest level of resistance and 9 = represents extreme susceptibility (presense of severe symptoms, damage, or stress). Plants were grouped into three depending on CBB scores. The three categories were: Resistant (score 1-3), intermediate (score 4-6) and susceptible (score 7-9).

The relationship between yield parameters and CBB severity in the F2 populations plants was determined through correlation coefficient (r) analysis (Gomez and Gomez, 1984). Analysis of variance and mean comparison of the CBB score was done using ReML. Entry means were saved along with the estimate of experimental error. These values were analyzed following the NCD2 procedure to estimate general combinig ability (GCA) and specific combining ability (SCA) values. The GCA and SCA values and variance components were determined to estimate the mode of gene action and heritability of CBB resistance.

The following model was used to determine the SCA:
Yijk = _… + GCAR + GCAS +SCAij + Bk + eijk
Thus SCAij = Yijk – (_… + GCAR + GCAS + Bk + eijk) i.e. SCAij = Observed - Predicted value

Where:

GCAR= General combining ability of the *CBB* resistant parent

GCAS = General combining ability of the *CBB* susceptible parent

SCAij = Specific combining ability of a cross between *CBB* resistant and susceptible parent

Bk =A constant

eijk = error

Heritability was estimated as the narrow sense coefficient of genetic determination (NS-CGD) and broad sense coefficient of genetic determination (BS-CGD).

The NS-CGD and BS-CGD were determined from the formulas:

$$\text{NS-CGD} = \frac{(\sigma^2GCA(R) + \sigma^2GCA(s))}{(\sigma^2GCA(R) + \sigma^2GCA(s) + \sigma^2SCA + eijk)}$$

$$\text{BS-CGD} = \frac{(\sigma^2GCA(R) + \sigma^2GCA(s) + \sigma^2SCA)}{(\sigma^2GCA(R) + \sigma^2GCA(s) + \sigma^2SCA + eijk)}$$

Baker's ratio was determined using the formula:

$$\text{Baker's ratio} = \frac{(\sigma^2GCA(R) + \sigma^2GCA(s))}{(\sigma^2GCA(R) + \sigma^2GCA(s) + \sigma^2SCA)}$$

Table 13: Skeleton ANOVA of North Carolina Design II matting design

Source	**d.f**	**MS**	**F cal**	**Fp**	**Exp MS**
Rep	1				
Crosses	11				
GCA(Resistant)	3		GCA(Resistant)/EMS		$\sigma^2_e+6\sigma^2_{GCA}(\text{Resistant})$
GCA(Susceptible)	3		GCA(Susceptible)/EMS		$\sigma^2_e+6\sigma^2_{GCA(}\text{Susceptible})$
SCA	5		SCA/EMS		$\sigma^2_e + 2\sigma^2_{SCA}$
Error					σ^2_e

4.2.3. Histogram and chi-square goodness-of-fit test for CBB F2 phenotypic classes

Histogram was used to determine the distribution of F2's in relation to CBB resistance (quantitative and qualitative). Chi-square test for goodness of fit was also used to determine the segregation pattern for CBB resistance in selected crosses.

A chi-square goodness-of-fit test was used to determine the deviation of the observed frequencies from the hypothesized frequencies, using;

$X^2 = \sum \frac{(obs - \exp)^2}{\exp}$ (Elrod and Stansfield,2002), where *Exp* is the expected count for a class and *Obs* is the count actually obtained. where χ^2 was significant at ($P < 0.05$), the fit of a model was rejected. Some of the most common phenotypic classes were tested: 3:1 (single dominant gene); 15:1 (duplicate dominant epistasis); 9:7 (duplicate recessive epistasis); 13:3 (dominant and recessive epistasis); 9:3:4 (Recessive epistasis); 9:6:1 (Duplicate gene with cumulative effect); 12:3:1 (Dominant Epistasis) (Elrod and Stansfield, 2002).

4.3. Results

The combining ability studies for CBB tolerance is summarized in Table 10. The ANOVA revealed that the GCA of the CBB susceptible (Female) parents were significantly ($P \leq 0.05$) different for final CBB means at (56 DAI), number of pods/plants at ($P \leq 0.001$), seed yield (weight in grams of total seed harvested per cross**)** at ($P \leq 0.05$) and weight of 100 seed at ($P \leq 0.05$). The GCA of the *CBB* resistant (Male) parents were not significantly different for any of the traits considered. SCA were not significantly different for CBB means at 56 DAI and seed size, but were significantly different for number of pods/plants at ($P \leq 0.001$) and weight of total seed harvested at ($P \leq 0.01$).

4.3.1. Inheritance of resistance to *CBB*

Analysis of variance showed that there was no significant difference in GCA effects among CBB resistant parents. However, the GCA effects of the CBB susceptible parents were significantly different ($P \leq 0.05$) (Table 14).

Table 14: Mean Squares of key traits used in analysis of inheritance study.

Source of variation	**Df**	**CBB means score (1-9) 56 DAI**	**No of pod/plant**	**Seed yield (gm)**	**100 seed weight (Seed size) (gm)**
GCA (S) Female	3	0.44*	1744.30***	464.5*	349.7 *
GCA (R) Male	3	0.21	14.90 ns	72.6 ns	107.61 ns
SCA	5	0.06	805.60 ***	730**	47.36 ns
Residual	11	0.084	43.30	86.55	34.19
σ^2 GCA(S)Female		0.118	567	125.98	105.17
σ^2 GCA(R)Male		0.041	-9.47	-4.65	24.48
σ^2 SCA		-0.022	762.31	643.45	6.59
σ^2 e’		0.084	43.30	86.55	126.73
Baker's Ratio		1	0.43	0.16	0.95
NS-CGD		0.65	0.41	0.15	0.49
BS-CGD		0.65	0.96	0.89	0.51

Abreviations: **R**=Resistant, **S**=Susceptible, *=Significant at 0.05, **=Significant at 0.01, ***=Significant at 0.001

Among the susceptible parents, K132 and Kanyebwa had negative GCA effects, suggesting that probably these genotypes had some level of resistance (or at least lesser susceptibility) and they may contribute to resistance in crosses involving them. One susceptible parent, K131 had the highest positive and significant ($P<0.01$) GCA effects for *CBB* score thus affirming its susceptibility to CBB and lack of any resistance genes to CBB (Table 15). Masindi yellow also

showed a low GCA effect value. This may imply that transferring resistance to K132 and Kanyebwa would probably be easier than to transferring it to K131 and Masindi Yellow. Also K131 and Masindi yellow would act as good susceptible checks in studies on CBB compared to Kanyebwa or K132.Among CBB resistant parental groups, MCM 2001 and RWV 2070 with negative GCA effects values (-0.102 and -0.281) had the most desired GCA effects and are therefore good combiners for CBB resistance. On the contrary, the GCA effect of MIB 456 and JESCA (CBB resistant parents) was relatively high for CBB score and therefore would probably be less effective at transferring resistance to its progeny.

Table 15. GCA effects of *CBB* resistant and susceptible parent

Group	**CBB means score 56 DAI (1-9)**	**No of pods/plant**	**Seed yield (gm)**	**100 seed weight (Seed size) (gm)**
Resistant				
MCM 2001	-0.102	4.07	16.4	-5.51
MIB 456	0.250	0.37	14.4	3.66
RWV 2070	-0.281	-2.13	8.8	-5.17
JESCA	0.194	-0.23	19.8	6.90
SE (Res)	**0.102**	**2.33**	**3.29**	**3.98**
Susceptible				
K 132 (CAL 96)	-0.263	-16.67**	44.2	-6.03
Kanyebwa	-0.252	-7.47	43.9	13.78
Masinde Yellow	-0.170	-16.67**	40.9	9.99
K 131	0.450**	28.73***	64.5	-9.85
SE (Susc)	**0.102**	**2.33**	**3.29**	**3.98**

*,** and *** refer to significant GCA effects at $P < 0.05$, $P < 0.01$ and $P < 0.001$ respectively, GCA without stars were non-significant at $P< 0.05$

The SCA effects of the CBB means at 56 DAI, were not significant. Baker's ratio for CBB means was 1.0 indicating that the genetic effects controlling *CBB* resistance were additive in the material used in this study. The narrow sense coefficient of genetic determination was 0.65 suggesting that 65% of the inheritance to *CBB* resistance is governed by additive genes.

The broad sense coefficient of genetic determination was also 0.65 indicating that 65% of the observed inheritance to *CBB* resistance is due to additive and non-additive gene effects and only 35% was due to error or environmental influence. The specific combining ability (SCA) effects of the crosses between the *CBB* resistant parent and *CBB* susceptible parents are also presented in Table 16. For *CBB* resistance determined in terms of percentage of leaves damaged per plants for each cross, a lower SCA value is desirable. The specific combining ability for CBB were not significant ($P<0.05$) for any crosses. The cross K 131 x MCM 2001 had the most desirable SCA (-0.24) followed by Masindi Yellow x RWV 2070 and K 132 x RWV2070 with SCA values of -0.18 and -0.14, respectively. The crosses K131 x RWV 2070 and Masindi Yellow x MCM 2001 had positive and undesirable SCA values, 0.34 and 0.24 respectively.

Table 16. Summary of SCA effects of the crosses

Crosses	**CBB means score at 56 DAI (1-9)**	**No of pods/plant**	**Seed yield (gm)**	**100 seed weight (Seed size) (gm)**
K 131 x Jesca	-0.05	6.13	15.24	-3.87
K 131 x MCM 2001	-0.24	-8.19	-3.55	4.01
K 131 x MIB 456	-0.05	0.06	-1.38	-4.81
K 131 x RWV 2070	0.34	2.00	-10.32	4.67
K 132 x Jesca	0.03	2.56	2.63	3.65
K 132 x MIB 456	0.11	31.50***	26.46*	5.37
K 132 x RWV 2070	-0.14	-34.06***	-29.08**	-9.02
Kanyebwa x Jesca	0.02	-8.69	-17.87	0.22
Kanyebwa x RWV 2070	-0.02	8.69	17.87	-0.22
Masinde Yellow X RWV 2070	-0.18	23.38**	21.53*	4.57

Masinde Yellow x MCM 2001	0.24	8.19	3.55	-4.01
Masinde Yellow x MIB 456	-0.06	-31.56***	-25.08*	-0.56
SE	**0.20**	**4.65**	**6.58**	**7.96**

,* and ******* refer to significant GCA effects at P < 0.05, P < 0.01 and P < 0.001 respectively, SCA without stars were non-significant at P< 0.05**.**

4.3.2 Inheritance of yield Parameters

The general combining ability (GCA) of the resistant groups were not significantly different for any of the three yield parameters under consideration. But for the susceptible group, they were significant different (P ≤0.05) for all the three yield parameters considered in this study. For number of pods, the Baker's ratio was (0.43), NS-CGD (0.41) and BS-CGD (0.43). For 100 seed weight the Baker's ratio was (1.00), NS-CGD (0.36) and BS-CGD (0.36). This implies a highly heritable trait associated with additive genes for 100 seed weight and low heritable trait for number of pods (Table 14). However, seed yield with Baker's ratio (0.16) and NS-CGD (0.15) were low although the BS-CGD (0.89) was high.

The susceptible parents K132 and Masindi yellow had a negative significant GCA effects for number of pods at P<0.01. Analysis further revealed that except for K131 with GCA effect for number of pods of (28.73), all the other *CBB* susceptible parents had unfavourable GCA effects for number of pods because they were negative. However MCM 2001 and MIB 456 resistant parents with (4.07) and (0.37) respectively had favourable GCA effects for number of pods. The GCA for seed yield was significant (P<0.001) for all the susceptible parents. Analysis also revealed that for seed yield all the susceptible parents had desirable GCA effects. but for 100 seed weight K132 and K131 had unfavourable GCA effects (Table 15).

The SCA for number of pods was significant (P<0.001). The cross K132 x MIB 456 had the most positive and significant SCA effect value for number of pods (31.50, P<0.001), followed by Masindi yellow x RWV 2070 (23.4) and significant at (P<0.01). The most negative were the crosses K 132 x RWV 2070 (-34.06) and Masindi yellow x MIB 456 (-31.56) and they were

significant at (P<0.001). There was significant (P<0.05) SCA effect for the cross K132 x MIB 456, with most positive SCA effect value (26.46) for seed yield. The crosses K 132 x RWV 2070 and Masindi yellow x MIB 456 having the most negative SCA effect values -29.08 and -25.08, were also significant at (P<0.01) and (P<0.05) respectively. The 100 seed weight was not significant (P>0.05) for all the parental crosses, the most positive cross was K 132 x MIB 456 (5.37) and the most negative SCA effects were crosses K 132 x RWV 2070 and K 131 x MIB 456 (-9.02) and (-4.81) (Table 16). In general, the results of this study showed that the inheritance of yield parameters were independent of the occurrence of CBB on beans.

4.3.3. Correlation analysis between CBB score at 56 DAI and yield parameters

The correlation for CBB severity and yield parameters showed that there was no statistically significant relationship (Table 17). The correlation coefficient indicated a relatively weak positive relationship between CBB severity and seed yield (r =0.36) and number of pods (r =0.51) as well. But the relationship with 100 seed weight was very low and negative (r = -0.067).

Table 17: Correlaction coefficients between CBB severity and yield parameters

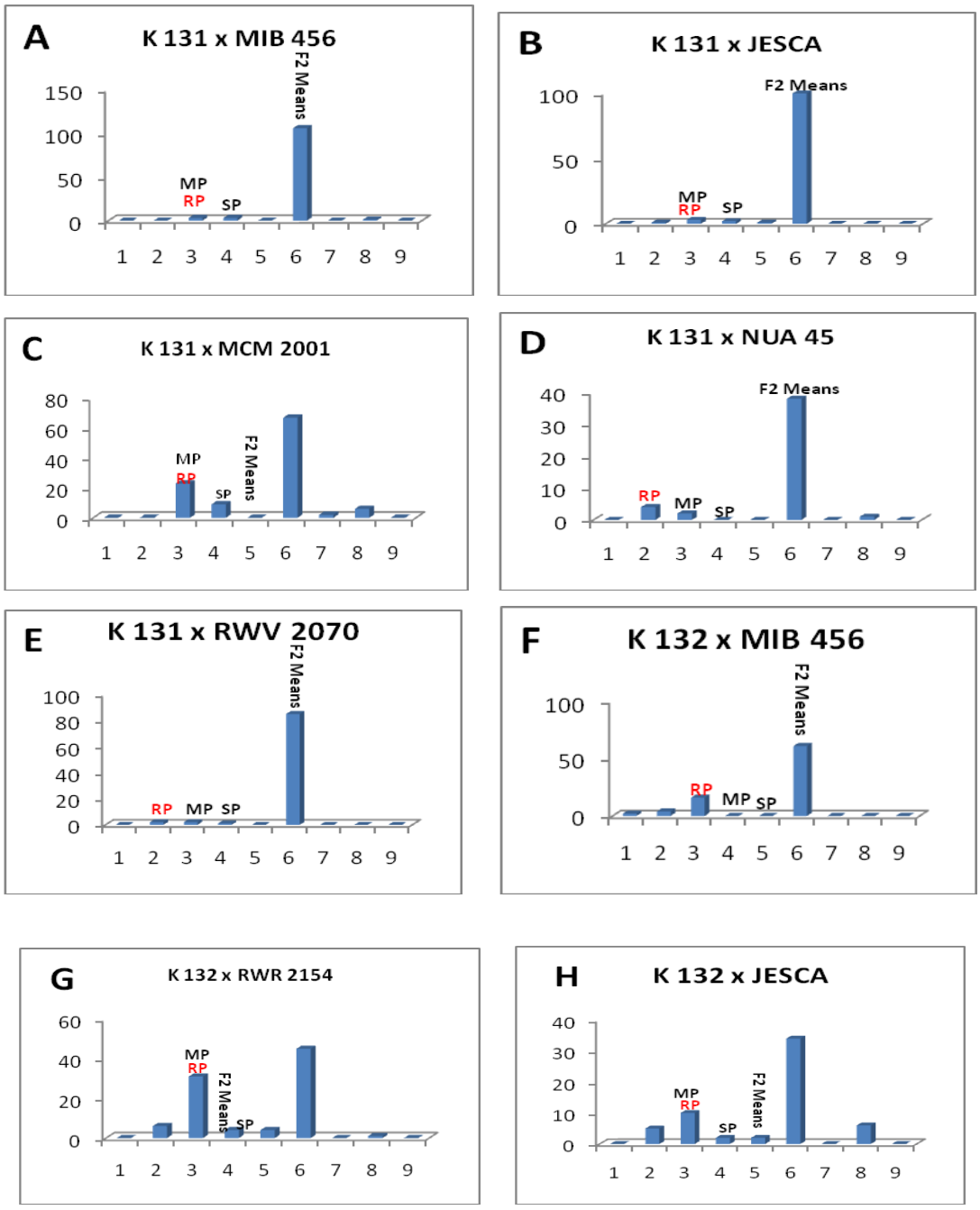
	CBB means score at 56 DAI	**N of pods/Plant**	**Seed yield**	**100 seed weight**
CBB means score at 56 DAI	1			
N of pods	0.328	1		
Seed yield	0.174	0.905***	1	
100 seed weight	-0.067	-0.202	-0.078	1

Abreviations: ***=Significant at (P≤0.001), correlaction values without stars were non-significant

4.3.3. Evaluation of CBB resistance and frequency distribution scores in F2 populations

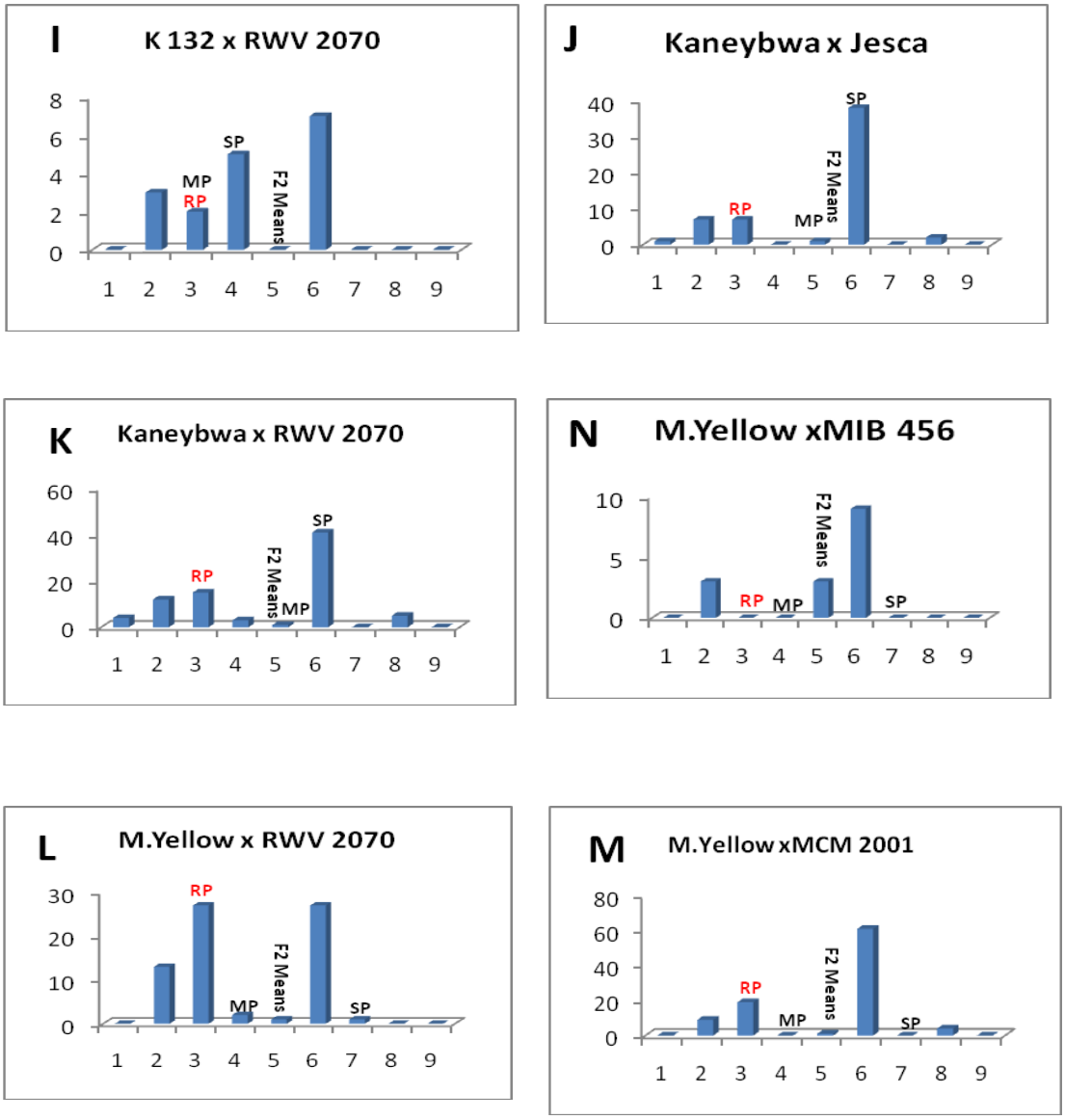
Mean severity scores indicated that the 14 families were segregating for CBB resistance with some progenies categorised as resistant, while others were moderately resistant or susceptible. (Table 18). The highest propotion of resistant plants was recorded from crosses of RWV 2070 with Masindi yellow (56%) and Kanyebwa (38%). Surprisingly, the least proportion of resistant plants (4%) was recorded in the cross K 131 x RWV 2070. The F2 distribuition histograms of CBB resistance for the 14 families are presented in figure 1. In general, mean CBB severity of the F2 populantions of the resistant by susceptible (R x S) crosses were less resistant than both resistant and susceptible parents and mid-parent as well.

The MP (Mid Parent) on six of the R x S crosses (Figure. 2 A, B, C, G, H and I) had the same value as the resistant parent (RP), on the rest of the crosses the mid parent was between RP and the susceptible parent (SP) or between RP and F2 progeny scores (Figure. 1 L, M and N). Six other R x S crosses (Figure 1 F, G, H, J, K and L) had skewed distributions towards the resistant parents. Whereas the cross Kanyebwa x Masindi yellow (Figure 1 K) had the same intermediate value for mid parent and F2 means as well (Figure 1 G) had the same intermediate value for SP and F2 means suggesting the presence of a dominant component for susceptibility.



CBB severity score (1-9)

Figure 1: Distribution frequency of CBB ratings for 14 F2 crosses from a 4 x 4 NCD2 evaluated on the Screenhouse CIAT-Uganda



***CBB* severity score (1-9)**

F2 mean=indicates the F2 Means , **MP** = Mid-Parent, **RP** = Resistant Parent, **SP** = Susceptible Parent

Figure 1: Distribution frequency of CBB ratings for 14 F2 crosses from a 4 x 4 NCD2 evaluated on the Screenhouse CIAT-Uganda

4.3.4. Segregartion Patterns of the F2 Populations

The pattern of segregation in F2 populations of CBB resistance for the 14 families is presented the Table 18. All the families tested for expected ratio 1R:3S significantly deviated from this expected phenotypic ratio for a single dominant resistance gene except the crosses K132 (S) x RWV 2070 (R) and K132 (S) X MIB456 (R). These crosses also fitted the expected ratio 3:13 ratio. This suggests the presence of at least one gene that showed dominance for susceptibiliy (Allard, 1999).

Table 18: Reaction and F2 means progenies for different CBB classes

Cross	**Resistant (1-3)**	**Moderatly resistant (4-6)**	**Susceptible (7-9)**	**Total plants per cross**
K131 x MCM 2001	23	71	13	**107**
M.Yellow x MCM 2001	28	59	7	**94**
K132 x RWR 2154	37	48	6	**91**
Kaneybwa x RWV 2070	31	39	11	**81**
K132 x RWV 2070	5	12	0	**17**
K132 x JESCA	15	38	6	**59**
M.Yellow x RWV 2070	40	28	3	**71**
K132 x MIB 456	22	61	0	**83**
K131 x MIB 456	3	102	8	**113**
M.Yellow x MIB 456	3	12	0	**15**
K131 x RWV 2070	4	86	0	**90**

K131 x NUA 45	6	35	4	**45**
K131 x JESCA	4	46	2	**52**
Kaneybwa x JESCA	15	37	4	**56**

Only K132 (S) x RWV 2070 (R) cross fitted a 7:9 phenotypic ratio, suggesting the involvement of two genes in duplicate recessive genes (Elrod & Stansfield, 2002). Four crosses; Masindi Yellow (S) x MCM2001 (R), K132 (S) x RWR2154 (R), Kaneybwa (S) x RWV2070 (R) and Masindi yellow (S) x RWV2070 (R), fit a phenotypic ration of 6:9:1 suggesting the presence of duplicate genes with cumulative effect (Fehr, 1987; Elrod and Stansfield, 2002). This implies that the gene controling resistance are recessive and can be contribuited by either parent or even both resistant and suspetible parent.

From the overall GCA effects of the parents KANYEBWA and K132, are susceptible, they had desirable (negative) GCA effects of -0.252 and -0.263 respectively, suggesting that they posses some levels of resistance, which can be transfered in a crosses were they are involved.

The crosses K131 (S) x MCM 2001 (R), K 132 (S) x JESCA (R), K132 (S) x NUA 45 (R), and KANYEBWA (S) x JESCA (R) matched on the 4:9:3 and 3:12:1 ratio suggesting that both recesive epistasis and dominant epistasis possible gene interactions were active in these crosses (Elrod and Stansfield, 2002). The cross K 132 (S) x JESCA (R) matched 3 ratios 6:9:1, 4:9:3, 3:12:1 suggesting that there are several plausible epistatic gene interactions associated with these crosses (Miko, 2008).

Table 19. Number of bean plants for F2 progenies, showing different levels of resistance

(Crosses)	Obs	No of plants	χ2 under different model ratios							
			1:3 (Prob)	**3:13 (Prob)**	**1:15 (Prob)**	**7:9 (Prob)**	**1:2:1**	**6:9:1 (Prob)**	**4:9:3 (Prob)**	**3:12:1 (Prob)**
K 131 x MCM 2001	23:71:13	107	-	-	-	-	23.13**	11.72**	11.93*	**0.91ns** (0.63)
M.Yellow xMCM 2001	28:59:7	94	-	-	-	-	21.83**	**3.66ns** (0.16)	12.97*	7.73***
K 132 x RWR 2154	37:48:6	91	-	-	-	-	30.96**	**4.17ns** (0.12)	24.11***	30.57***
Kanyebwa x RWV 2070	31:39:11	84	-	-	-	-	18.58**	**0.14ns** (0.93)	12.11***	18.35***
K 132 x RWV 2070	5:12	17	**0.18ns** (0.67)	**1.27ns** (0.26)	15.56***	**1.42ns** (0.23)	-	-	-	-
K 132 x JESCA	15:38:6	59	-	-	-	-	7.64*	**4.44ns** (0.11)	**3.02ns** (0.22)	**3.73ns** (0.15)
M.Yellow x RWV 2070	40:28:3	71	-	-	-	-	41.73**	3.09 *	19.84***	65.94***
K 132 x MIB 456	22:61	83	**0.10ns** (0.75)	**3.28ns** (0.07)	58.12***	10.03**		-	-	-

	3:109:						97.64*			
K 131 x MIB 456	1	113	-	-	-	-	**	74.27***	462.58***	27.76***
M.Yellow xMIB			**0.20ns**	**0.02ns**						
456	3:12	15	(0.65)	(0.90)	4.84*	3.44**		-	-	-
K 131 x RWV 2070	4:86	90	20.28**	12.09***	**0.50ns** (0.48)	56.50**		-	-	-
							189.95			**2.41ns**
K131 x NUA 45	6:35:4	45	-	-	-	-	***	14.54***	15.37***	(0.30)
							102.21			
K 131 x JESCA	4:46:2	52	-	-	-	-	***	69.66***	69.86***	30.76***
	15:37:						147.00			**2.79ns**
Kanyebwa x Jesca	4	56	-	-	-	-	***	**4.14ns** (0.13)	8.74**	(0.25)

, **, ** significant deviation from model ratios at 0.05, 0.01, 0.001 probability; **ns**=no significant deviation from model ratios; **Obs** = observed ratio, - =have not been tested on this model from the observed ratio

4.4. Discussion

4.4.1. Inheritance of resistance to CBB

Differences among the GCA effects of the genotypes suggest additive gene effects for CBB infection. The lower GCA values correspond to superior parents and indicate greater CBB resistance (Rosana Rodrigues *et al.,* 1999). This supports the findings of this study where the susceptible parent K131 had positive CBB average score GCA effects thus confirming its susceptibility to CBB infection. Among *CBB* resistant parental groups, MCM 2001 and RWV 2070 had the most desired GCA effects and can therefore be considered good combiners for CBB resistance. On the other hand, K131 and Masindi yellow (*CBB* susceptible parents) and MIB 456 (*CBB* resistant parent) had high GCA values for CBB resistance, implying that they are less desirable for breeding purposes in comparison to the other parents. Parents with the best (negative) GCA effects are potentially superior and may be included in breeding programs to select new inbred lines in advanced generations (Ramalho *et al.,* 1993).

Baker's ratio for *CBB* means score at 56 DAI was (1.0), implying that additive genes effects were more important than non additive gene effects (Rosana Rodrigues *et al.,* 1999). Thus, it would be easy to select for this trait phenotypically, because Baker's ratio is an indication of how progenies performance can be predicted from the GCA values of the parents. Therefore, since the progenies performance can be predictable, fewer crosses are needed to be made. The narrow sense coefficient of genetic determination was estimated as 0.65 suggesting that 65% of the inheritance to CBB resistance was governed by additive genes, a fact which is confirmed by the high Baker's ratio. The broad sense coefficient of genetic determination is 0.65 indicating that 65% of the observed inheritance to CBB resistance is due to additive and non-additive gene effects and only 35% was due to error or environmental influence, implying that their inheritance for this trait was largely controlled by genotypic effects and less affected by environmental factors. For this particular variable, the broad sense coefficient of genetic determination and narrow sense coefficient of genetic determination had the same estimation value of 0.65 because the SCA variance estimate its true value is negative (Table 14). The results indicate a real

possibility of greater efficiency in the selection for CBB resistant individuals in this population. From the cross between VAX 4 one of the VAX's lines, known to be CBB resistant and Kablanketi (susceptible), was estimated moderate heritability of 0.32 implying that resistance is conditioned by one major gene which has effects of particial resistance (George *et al.*, 2012).

Heritabilty in CBB resistance has been widely studied, however with certain differences between the results (Claudia *et al.*, 2003). *et al.*, (1989), reported that additive gene action was significant, with heritability estimates in narrow sense ranging from 0.18 to 0.87. Ariyarathne *et al.*, (1998) reported heritability estimates in narrow sense ranging between 0.30 to 0.60, Arnaud-Santana *et al.*, (1994) reported 0.52 to 0.60 and Singh *et al.*, (2001) 0.09 to 0.93. In general, the heritability values depend on several aspects such as the population into consideration environmental conditions, the experimental design, the accuracy of the data collection and more importantly, the genetic complexity of this trait (Claudia *et al.*, 2004). Therefore, the differences in the results of heritability for CBB resistance are quite common, thereby emphasizing that there is still much research to be done in relation to the complexity of this disease (Claudia *et al.*, 2004). In general the results in this study indicates that additive genes effects were more important than non additive gene effects, implying that the progenies performance can be predicted from the GCA values of the parents.

4.4.2. Evaluation of CBB resistance and frequency distribution scores in F2 populations

The mean phenotypic score for the 14 studied families was intermediate signifying continual segregation of the progeny (Kachulu, 2011). But the individual scores for the progeny within six of the populations revealed presence of resistant genotypes in the F2 populations. Six other R x S crosses (Figure 1 F, G, H, J, K and L) had skewed distributions towards the resistant parents. The cross represented in Figure 1 K had the same intermediate scoring value for mid parent and F2 means while that represented in Figure 1 G had the same intermediate value for SP and F2 means, suggesting the presence of a dominant component for susceptibility (Bolek *et al.*, 2005).

On the presence of dominance effects, the segregating populations in advanced generations of hybrids tends to resemble one parental phenotype more than the other. But some unpredictable deviation from the additive and dominance expectation can occur when additional epistatic effects are present (Wijngaarden and Brakefield, 2000; Schluter *et al.*, 2004; Kearsey and Pooni, 1998). This probably explains some of the unpredictable distributions among the R x S crosses in this study generally suggesting some possible form of epistasis. Most of the F2 populations distribution of resistant by susceptible (R x S) crosses had distinct phenotypic classes with some shifting towards resistance, implying that CBB resistance is most likely quantitatively inherited in these crosses (Bonos, 2006). In this study, all the frequency distribution of the F2‘s from R x S crosses presented a wide and skewed distribution. This may be due not only to dominance, but also to epistasis which causes deviations from the expected distribution (Griffith *et al*., 1997). Even though most F2 populations had continuous distribution, a range of different segregation patterns are observed suggesting that inheritance of resistance to CBB was complex.

4.4.3 Segregation patterns of the F2 Populations

The pattern of segregation in the F2 populations of the 14 studied families showed that the mode of gene action governing CBB resistance varied depending on the parents involved. The crosses K132 (S) x RWV 2070 (R) and K132 (S) X MIB 456 (R) fitted on the ratio 3:1 and 3:13 as well suggesting the presence of at least one gene that showed dominance and making both a single gene and a two gene explanation plausible (Table 19)

The cross K131(S) x RWV 2070 (R) is the only are which fitted the ratio 1:15 suggesting the presence of duplicate dominant genes, where the presence of one dominant allele from both or one locus is epistatic to either recessive condition (Elrod and Stansfield, 2002). This implies that the genes conditioning resistance in MCM 2001 are recessive. The crosses Masindi Yellow (S) x MCM 2001 (R), K 132 (S) x RWR 2154 (R), KANYEBWA (S) x RWV 2070 (R), K 132 (S) x JESCA (R), Masindi Yellow (S) x RWV 2070 (R) and KANYEBWA (S) x JESCA (R) matched on the 6:9:1. (Table 19). Ratio suggesting presence of duplicate genes with cumulative effect, where the dominant condition (homozygous or heterozygous) at only one of the loci produces the same phenotype (Elrod and Stansfield, 2002). It implies that possibly the genes on the resistant

parents, MCM 2001, RWV 2154, RWV 2070 and JESCA are recessive resistant. The cross K 132 (S) x JESCA (R) and KANYEBWA (S) x JESCA (R) also matched the ratios; 4:9:3, 3:12:1. (Table 19).

Ratios suggesting that both of these ratios can plausibly explain the gene action in this cross (Miko, 2008). The ratio 4:9:3 suggests the presence of recessive epistasis, where the recessive genotype at one locus suppresses the expression of alleles on the other locus, in which case the first locus is said to exhibit recessive epistasis over the second locus. The ratio 3:12:1 suggests dominant epistasis, in which a dominant allele at one locus is able to express itself producing a certain phenotype regardless of the allelic condition of the other locus. In this study, in both situations resistance was expressed in homozygous recessive condition, confirming that the genes conferring resistance on the parent JESCA are recessive.

On the other hand, Muimui *et al*, (2011), using some of the known sources of resistance from VAX's lines and Wilk line, found that the segregation of the F2 generation for resistance/susceptibility to *Xcp* in the crosses Lusaka Yellow x Wilk 2, Lusaka Yellow x VAX 6, Pembela x Wilk 2 and Pembela x VAX 6 did not differ from the expected 3:1 ratio indicating that resistance to common bacterial blight in Wilk 2 and VAX 6 could be governed by a single dominant gene. George *et al.* (2012) reported crosses between the susceptible parent Kablanketi and the resistant parent VAX 4 in which their F1, F2 and the backcrosses to both parents were generated. They found that there was no significant deviation from the expected 3:1 ($\chi 2= 0.47$; $P>0.05$) in the F2 population and 1:1 for the backcross to the susceptible parent, suggesting that resistance in VAX 4 to Xcp was conditioned by the presence of dominant genes.

The finding in this study also suggests that genetic resistance to *Xcp* in common bean genotypes is controlled by more than one gene with varying degrees of gene action (Chataika, 2011). These findings are similar to those reported by several authors that have reported *Xcp* resistance to be controlled by one or more genes (Beebe and Pastor-Corrales, 1991; Zapata *et al*., 2009). From the crosses between PR0313-58 (resistant) x Rosa Nativa (susceptible), Zapata *et al.* (2010) were the first ever to report a single gene conferring resistance to *Xcp* in common bean. They considered that resistance to *Xcp* strain 3353 was conferred by a single dominant gene. The new sources of resistance MCM 2001, MIB 456, NUA 45, JESCA, RWR 2154 and RWV 2070 identified in this

study appeared to have more than one gene with resistance expressed in a recessive allelic condition.

In general the inheritance and gene action to *Xcp* among other factors is influenced by plant architecture (Beebe and Pastor-Corrales, 1991). The type IV climbing bean with vigorous vegetative growth usually grows on stakes spreading its canopy in air. These beans therefore do not allow moisture to accumulate around leaves and thus do not favour the development of CBB. On the other hand the bush types' canopy which is crowded and close to the ground experiences a different microclimate that has more moisture. In these two architectural types, resistance to CBB is expressed differently.

In this study, the F2 distribution analysis and the chi-square test suggest the presence of at least two or three genes with interactive effects. It also shows that the resistance in the new identified sources is conditioned by recessive genes. However, dissection of a truly quantitative variation into its underlying Mendelian factors is difficult to achieve only from phenotypic information, requiring a molecular technique to answer the question of number of genes and size of effects (Jansen, 2001).

4.4.4. Inheritance of yield components

Significant differences were shown by the mean squares for number of pods, seed yield and 100 seed weight suggesting that large genetic differences exist among the genotypes for these yield components. Desirable parents would be those with significant GCA effects in the right direction (desirable effect) for the trait of interest (Singh and Chaundary, 2004). The right direction for the yield parameters is positive GCA values. The high positive GCA effects observed for number of pods per plant on the parent K 131 (28.7), implies that this parent possess favourable alleles for this trait which is an important character in breeding for yield on CBB resistant genotypes. SCA refers to the performance of the parental combinations compared to the value predicted by the GCA values of the parents involved. For example, a cross between K 132 (S) x MIB 456 (R) which had a desirable SCA effect for all the three yield parameters under analysis: number of

pods, seed yield and 100 seed weight, with SCA effects values of 31.5, 26.46 and 5.37 respectively. Although the cross did not have good SCA effects (0.11) for CBB resistance, it can be exploited to generate progenies with good yield performance. However the low Baker's ratio (0.16) and NS-CGD (0.15) for seed yield revealed low predictability and heritability of this trait and also the predominance of non-additive effects.

The BS-CGD value (0.89) indicates that 89% of the phenotypic variation is due to additive and non-additive genetic effects, but only 15% from NS-CGD can be heritable. This suggests that additive effects did not have much role in controlling inheritance of this particular trait (Alghamdi, 2009). On the other hand the number of pods had high Baker's ratio (0.43), NS-CGD (0.41) and BS-CGD (0.96) suggesting that the inheritance of yield components is simpler and highly heritable than the actual production of seed (Checa and Blair, 2012).

The F2 progenies had a wide range of seed size which varied from 13gm to 77gm. This could be attributed to segregation of genes controlling seed size (Upadhyaya *et al*., 2006). Baker's ratio (1.00), NS-CGD (0.36) and BS-CGD (0.36) showed that 100 seed weight was largely controlled by non additive genes (Table 17). Cho *et al*. (2002) also reported that quantitative trait loci (QTL) accounted for 52% of total phenotypic variation for seed size in chickpea. The results also show that yield in beans like any other crop is a complex character where many morphological and physiological characteristics influence it. These yield contributing characters are also reported to be interrelated in complex relationships (Raffi and Nath, 2004). The crosses K132 (S) x JESCA (R), K132 (s) x MIB 456 (R) and Masindi yellow x RWR 2070 had good mean values for all the yield parameters and were also good combiners for yield. In general, the results of this study showed that the inheritance of yield parameters are independent of the occurrence of CBB on beans. It was however clear that the occurrence of CBB particularly in susceptible cultivars drastically reduces the yield and seed quality. Fininsa (2003) reported about 5.2 and 9.1 kg/ha beans yield loss in pure stand and bean-maize intercropping systems respectively for each per cent increase of CBB severity in broadcast and mixed intercropping. On the other hand the growth habit of bean has great influence on yield.

4.4.5. Correlation analysis between CBB score at 56 DAI and yield parameters

The correlation coefficient indicated a relatively positive relationship between CBB score and seed yield (r =0.174) and number of pods (r =0.328). These results suggest that CBB on the screen house did not have very significant effect on yield parameters. This correlation can be due to several causes: CBB may not affect as such, but may affect the quality of seed. So that if we consider the yield of "clean and marketable seed" most likely the correlation would have been negatively stronger. Scott and Michaels, (1992) reported a significant ($P < 0.05$) strong positive correlation of 0.72 between severity of blight and yield loss when evaluating *Xanthomonas* resistance of *phaseolus* interspecific cross selections confirmed by field performance or perhaps less likely, among others factores, CBB infection occurs most readily where there are wind-driven rains (Schwartz *et al.,* 2007). Probably these conditions did not exist on the screen house, therefore explaining the lack high correlation between CBB severity and yield.

4.5. Sectional conclusion and recommendations

This chapter mainly addressed the mode of inheritance and gene action for CBB resistance. This was achieved by crossing six exotic genotypes identified on the Chapter III of this thesis as CBB resistant with four local preferred and adapted genotypes. North Carolina II (NCD2) mating design was used to develop the study populations. Results revealed that both additive and non additive gene effects were involved in controlling resistance for CBB on beans. However, based on the Baker's ratio, additive effects were found to be more important. The genotypes MCM 2001 and RWV 2070 were found to have the most desirable GCA among the six resistant parents. The genotype MCM 2001 had also desirable GCA to some yield parameters such as number of pods and seed yield. These parental genotypes thus can be exploited for both CBB resistance and increase yield as well.

CHAPTER FIVE

GENERAL DISCUSSION, CONCLUSION AND RECOMENDATIONS

5.1. General discussion

Since the identification of CBB as an important bean disease in Uganda in 1960 (Leakey, 1963), no particular attention was given to it until 1983 where research was initiated on the disease. Since then considerable effort has been devoted to understanding the disease and its causal organism and developing resistant genotype to the disease (Opio and Namayanja, 2002). Breeding particularly focused on improving locally accepted varieties for resistance to CBB. Unfortunately, most of generated materials have never been released because they lacked other farmer desirable attributes hence susceptible but farmer preferred varieties have continued to dominate. This study was conducted to generate knowledge and thus contribute to the improvement of common beans production in Uganda through identifying and characterizing effective resistance genes to CBB in Uganda and determining the mode of inheritance of this resistance in varieties preferred and grown by farmers.

The first study was aimed at identifying new sources of resistance to *Xcp* and to identify possible CBB resistant parents for use in improving locally adapted common bean varieties in Uganda. Two screening trials were conducted under green house conditions and artificial inoculation. The results revealed that final CBB mean scores ranged between 1 to 8 on a 1-9 scale in both screening cycles, implying that the genotypes ranged from resistant to highly susceptible.

After two rounds of screening, 10 lines were considered resistant. The genotypes, RWV 2070, RWR 2154, MCM 2001, NUA 45, JESCA and MIB 456 were selected resistant. Their resistance was comparable to that in VAX's lines (Vax 1, Vax 2, Vax 3, Vax 4, Vax 5 and Vax 6) that are used as a source of CBB resistance (Singh and Muñoz, 1999).

The second study was aimed at determining the mode of inheritance of resistance to CBB on common beans. The results revealed that both additive and non-additive gene effects were

involved in controlling resistance to CBB on beans. There was evidence of minor resistance genes in two of the susceptible parents (K132 and Kanyebwa) that are popularly grown in Uganda. This could probably explain their survival and long existence as cultivars of choice under farmers' conditions. However, based on the Baker's ratio, additive effects were found to be more influential in determining resistance to CBB than non-additive gene affects. The results also revealed from the segregation patterns that more than one gene was involved in conferring resistance to CBB, suggesting dominance and some epistatic interactions. In terms of selection strategy, these findings suggest that the single-seed-descent method or F2-derived families harvested in bulk can be more appropriate to breed for CBB resistance as both methods can be conducted out of the germplasm adaptation region. This allows for maximum expression of the genetic variance among lines in the final population. It is therefore a great alternative, especially when screen house is available for advancing generations. Since it is not influenced by the environment, it is possible to advance two to three generations per year (Borém and Miranda, 2009). Moreover, it allows generations of advanced lines without loss of alleles per selection because the original variability is maintained until late generations (Allard, 1971).
Additionally, selection for high heritability characters can be practiced in individual plants (Borém and Miranda, 2009).

5.2 Conclusion

From this study six genotypes namely JESCA, RWV 2070, RWR 2154, MIB 456, NUA 45 and MCM 2001 were found to be resistant to CBB. These could probably replace the exotic sources of resistance that have for a long time been used in most African CBB breeding programs. Utilizing adapted germplasm as sources of resistance would result in better breeding progress than utilizing exotic sources.

Effective sources of resistance among the newly identified parents were determined based on the gene action of the resistance they bear. These were considered suitable for breeding for CBB resistance. They included; MCM 2001 and RWV 2070 based on the largest negative GCA effects. The positive GCA and SCA were desirable for yield parameters such as number of pods per plant, seed yield and seed size. The genotype MCM 2001 also had desirable GCA effects for some yield

parameters such as number of pods and seed yield. The breeding implication from this is that parents with desirable GCA and SCA effects should be used for a specific trait. The genetics studies revealed that inheritance of CBB resistance in beans is controlled by additive and non-additive gene actions though the former was found to be more important. This implied that single-seed-descent method or F2-derived families harvested in bulk which are both convenient for high hereditability estimation values, can be appropriated as a selection method.

5.3. Recomendations

Based on the results of the study, the following recommendations can be made:

i) The resistant parental genotypes MCM 2001 and RWV 2070 and susceptible parental genotype KANYEBWA displayed high negative GCA effects for CBB resistance. Even though they did not have the best GCA effects for the yield parameters, they can be used for improving CBB resistance in the preferred beans in Uganda.

ii) Among various factors, the inheritance of resistance to CBB is highly dependent on the material used as resistant or susceptible parent. Therefore, inheritance of resistance to CBB should be investigated in a wide range of parental sources. Since additive gene action was more important than non-additive gene effects, breeding methods such as crossing and selfing or backcrossing that make the best use of additive variance, should be used to transfer CBB resistance into susceptible commercial and preferred varieties.

iii) Generations could also be advanced by the single-seed-descent method or F2-derived families harvested in bulk. Seed should be multiplied while their performance is tested against their parents in on-station trials under field conditions. Later the promising genotypes should be subjected to multi-locational trials to test stability of their performance while enriching findings regarding their gene action. The promising stable varieties identified should be subjected to selection.

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Appendices

Appendix I. Means separations for the variable under analysis for the ACC lines

14 DAI			35 DAI			AUDPC			r_AUDPC		
ACC 10	2.50	A	ACC 22	2.00	A	ACC 18	43.80	a	ACC 18	0.26	a
ACC 18	2.50	A	ACC 1	2.50	Ab	ACC 22	45.00	ab	ACC 22	0.26	ab
VAX 6	2.50	A	ACC 10	2.50	Ab	VAX 6	45.00	ab	VAX 6	0.26	ab
ACC 17	3.00	Ab	ACC 14	2.50	Ab	ACC 10	46.30	abc	ACC 10	0.27	abc
ACC 21	3.00	Ab	ACC 18	2.50	Ab	ACC 17	48.50	a-d	ACC 21	0.28	a-d
ACC 22	3.00	Ab	ACC 24	2.50	Ab	ACC 21	48.50	a-d	ACC 4	0.28	a-d
ACC 3	3.00	Ab	ACC 3	2.50	Ab	ACC 4	48.50	a-d	ACC 17	0.28	a-d
ACC 4	3.00	Ab	ACC 4	2.50	ab	ACC 3	49.60	a-e	ACC 3	0.29	a-e
ACC 5	3.00	Ab	ACC 5	2.50	ab	ACC 5	49.60	a-e	ACC 5	0.29	a-e
MCM2001	3.00	Ab	ACC 6	2.50	ab	MCM2001	49.60	a-e	MCM2001	0.29	a-e
ACC 1	3.50	a-c	ACC 8	2.50	ab	ACC 8	52.00	a-e	ACC 8	0.30	a-e
ACC 16	3.50	a-c	MCM2001	2.50	ab	ACC 24	53.30	a-e	ACC 24	0.31	a-e
ACC 24	3.50	a-c	VAX 3	2.50	ab	ACC 14	54.30	a-e	ACC 14	0.32	a-e
ACC 8	3.50	a-c	ACC 17	2.50	ab	ACC 1	54.50	a-e	ACC 1	0.32	a-e
ACC 14	4.00	a-d	ACC 21	2.50	ab	VAX 3	55.50	a-e	VAX 3	0.33	a-e
VAX 3	4.00	a-d	VAX 6	2.50	ab	ACC 16	56.80	a-e	ACC 16	0.33	a-e

ACC 15	4.50	a-e	ACC 15	3.00	abc	ACC 6	60.30	a-f	ACC 6	0.35	a-f
ACC 19	4.50	a-e	ACC 16	3.00	abc	ACC 19	65.00	a-f	ACC 19	0.38	a-f
ACC 2	4.50	a-e	ACC 19	3.00	abc	ACC 15	66.30	a-g	ACC 15	0.39	a-g
ACC 6	4.50	a-e	ACC 23	3.00	abc	ACC 2	69.80	a-h	ACC 2	0.41	a-h
ACC 12	5.00	a-f	ACC 2	3.50	a-d	ACC 23	72.00	a-h	ACC 23	0.42	a-h
ACC 20	5.50	b-g	ACC 20	3.50	a-d	ACC 12	75.50	a-i	ACC 12	0.44	a-i
ACC 23	5.50	b-g	ACC 9	3.50	a-d	ACC 20	76.80	a-i	ACC 20	0.45	a-i
ACC 9	6.00	c-g	VAX 1	3.50	a-d	ACC 9	79.00	b-i	ACC 9	0.46	b-i
VAX 1	6.00	c-g	VAX 2	3.50	a-d	VAX 2	80.30	c-i	VAX 2	0.47	c-i
VAX 2	6.00	c-g	VAX 5	3.50	a-d	VAX 1	81.50	d-i	VAX 1	0.48	d-i
ACC 13	6.50	d-g	ACC 12	4.00	b-d	VAX 5	83.80	e-i	VAX 5	0.49	e-i
VAX 5	6.50	d-g	VAx 4	4.00	b-d	VAx 4	92.00	f-i	VAx 4	0.54	f-i
ACC 11	7.00	e-g	ACC 13	4.50	cd	ACC 13	93.30	f-i	ACC 13	0.55	f-i
ACC 7	7.00	Efg	Kanyebwa	4.50	cd	Kanyebwa	100.25	ghi	Kanyebwa	0.57	ghi
VAx 4	7.00	Efg	ACC 11	5.00	d	ACC 11	101.50	hi	ACC 11	0.59	hi
Kanyebwa	7.50	Fg	ACC 7	5.00	d	ACC 7	102.80	hi	ACC 7	0.60	hi
M Yellow	8.00	G	M Yellow	5.00	d	M Yellow	108.50	h	M Yellow	0.64	i

NB: Means with the same letters are not statistically different

Appendix 2. Means separation for CBB scores on First screening

First Screening			
CBB score at 35 DAI	CBB score at 56 DAI	AUDPC	r-AUDPC
RWR 3006	CAB2	RWR 3006	RWR 3006
2.5 a	3.0 a	114.0 a	0.28 a
Zebra	CODMLB 003	Zebra	Zebra
2.5 a	3.0 a	124.5 a	0.30 ab
AFR 708	Gitanga 1	KAT 39	KAT 39
3.0 ab	3.0 a	125.5 abc	0.31 abc
CAB2	KAT 31	MAHARAJI SOJA	MAHARAJI SOJA
3.0 ab	3.0 a	130.8 a-d	0.32 a-d
CAL 143	KAT 39	CAB2	CAB2
3.0 ab	3.0 a	131.8 a-e	0.32 a-e
CODMLB 003	MCM 2001	Gitanga 1	Gitanga 1
3.0 ab	3.0 a	131.8 a-e	0.32 a-e
GASIRIDA	MIB 456	KAT 31	KAT 31
3.0 ab	3.0 a	131.8 a-e	0.32 a-e
Gitanga 1	MONTALCOM	UDSK-CBB-15	UDSK-CBB-15
3.0 ab	3.0 a	131.8 a-e	0.32 a-e
JESCA	RW 547	ROBA 1	ROBA 1
3.0 ab	3.0 a	136.8 a-e	0.32 a-e
KAT 31	RWV 2070	Nain de Kyondo	Nain de Kyondo
3.0 ab	3.0 a	137.0 a-f	0.33 a-f
KAT 39	RWV 2245	CODMLB 003	CODMLB 003
3.0 ab	3.0 a	138.0 a-f	0.33 a-f
MAC 44	RWR 3006	JESCA	JESCA
3.0 ab	3.0 a	138.0 a-f	0.33 a-f

MAHARAJI SOJA	SMC 16	MCM 2001	MCM 2001
3.0 ab	3.0 a	138.0 a-f	0.33 a-f
MCM 2001	SMC 21	MIB 456	MIB 456
3.0 ab	3.0 a	138.0 a-f	0.33 a-f
MIB 456	UDSK-CBB-15	MONTALCOM	MONTALCOM
3.0 ab	3.0 a	138.0 a-f	0.33 a-f
MONTALCOM	ACC 714	RW 547	RW 547
3.0 ab	3.5 ab	138.0 a-f	0.33 a-f
NABE 3	CAL 143	RWV 2070	RWV 2070
3.0 ab	3.5 ab	138.0 a-f	0.33 a-f
Nain de Kyondo	Nain de Kyondo	RWV 2245	RWV 2245
3.0 ab	3.5 ab	138.0 a-f	0.33 a-f
NGWINXCAB2	NGWINXCAB2/	SMC 16	SMC 16
3.0 ab	3.5 ab	138.0 a-f	0.33 a-f
ROBA 1	RWR 719	SMC 21	SMC 21
3.0 ab	3.5 ab	138.0 a-f	0.33 a-f
RW 547	SMC 17	NABE 3	NABE 3
3.0 ab	3.5 ab	142.2 a-f	0.34 a-g
RWR 719	VAX 2	CAL 143	CAL 143
3.0 ab	3.5 ab	143.2 a-h	0.34 a-h
RWV 2070	MAHARAJI SOJA	MAC 44	MAC 44
3.0 ab	3.5 ab	143.2 a-h	0.34 a-h
RWV 2245	KAB06F2.8-12	NGWINXCAB2	NGWINXCAB2
3.0 ab	3.5 ab	143.2 a-h	0.34 a-h
SMC 16	KAT 69	RWR 719	RWR 719
3.0 ab	3.5 ab	143.2 a-h	0.34 a-h
SMC 17	MAC 44	SMC 17	SMC 17
3.0 ab	3.5 ab	143.2 a-h	0.34 a-h
SMC 21	AFR 708	VAX 2	VAX 2
3.0 ab	4.0 abc	143.2 a-h	0.34 a-h
UDSK-CBB-15	DOR 500	GASIRIDA	GASIRIDA

3.0 ab		4.0 abc		147.5 a-i		0.35 a-i	
VAX	2	HM	21-7	AFR	708	AFR	708
3.0 ab		4.0 abc		148.5 a-j		0.35 a-j	
ACC	714	NABE	3	RW	1180	RW	1180
3.5 abc		4.0 abc		153.8 a-j		0.37 a-j	
CAL	96	ROBA	1	ACC	714	ACC	714
3.5 abc		4.0 abc		154.8 a-j		0.37 a-j	
DECELEYA	1	RW	1180	KAB06F2.8-12		KAB06F2.8-12	
3.5 abc		4.0 abc		154.8 a-j		0.37 a-j	
DOR	500	RW	184	KAT	69	KAT	69
3.5 abc		4.0 abc		154.8 a-j		0.37 a-j	
GURUKURARE		RWV	2154	HM	21-7	HM	21-7
3.5 abc		4.0 abc		160.0 a-k		0.38 a-k	
HM	21-7	Zebra		RW	184	RW	184
3.5 abc		4.0 abc		160.0 a-k		0.38 a-k	
KAB06F2.8-12		DECELEYA		RWR	2154	RWR	2154
3.5 abc		4.5 a-d		160.0 a-k		0.38 a-k	
KAT	69	GURUKURARE		NUV	219-1	NUV	219-1
3.5 abc		4.5 a-d		164.2 a-l		0.39 a-l	
KIVUZU		GASIRIDA		DECELAYA	1	DECELAYA	1
3.5 abc		4.5 a-d		165.2 a-m		0.39 a-m	
MAC	74	KIVUZU		GARUKURARE		GARUKURARE	
3.5 abc		4.5 a-d		165.2 a-m		0.39 a-m	
Masindi	Yellow	MAC	42	KIVUZU		KIVUZU	
3.5 abc		4.5 a-d		165.2 a-m		0.39 a-m	
MCB	49-89A	NUA	45	NUA	45	NUA	45
3.5 abc		4.5 a-d		165.2 a-m		0.39 a-m	
Ngakwu-Ngakwu		NUA59		DOR	500	DOR	500
3.5 abc		4.5 a-d		166.2 a-m		0.40 a-m	
NUA	45	VRA	4	MAC	74	MAC	74
3.5 abc		4.5 a-d		169.5 a-m		0.40 a-m	

NUV 219-1	AGRONOME	Masindi Yellow	Masindi Yellow
3.5 abc	5.0 b-e	170.5 a-m	0.41 a-m
RW 1180	KAB06F2.8-27	RW 805	RW 805
3.5 abc	5.0 b-e	170.5 a-m	0.41 a-m
RW 184	KAT 56	SMC 18	SMC 18
3.5 abc	5.0 b-e	170.5 a-m	0.41 a-m
RW 805	Masindi Yellow	VCB 81013	VCB 81013
3.5 abc	5.0 b-e	170.5 a-m	0.41 a-m
RWV 2154	MCB 49-89A	CAL 96	CAL 96
3.5 abc	5.0 b-e	175.8 b-m	0.42 b-m
SMC 18	NUV 219-1	KAB06F2.8-27	KAB06F2.8-27
3.5 abc	5.0 b-e	175.8 b-m	0.42 b-m
VAX 4	RW 582	MAC 42	MAC 42
3.5 abc	5.0 b-e	176.8 b-m	0.42 b-m
VCB 81013	RW 805	MCB 49-89A	MCB 49-89A
3.5 abc	5.0 b-e	176.8 b-m	0.42 b-m
GLP 2	SMC 18	VAX 4	VAX 4
4.0 bcd	5.0 b-e	176.8 b-m	0.42 b-m
ICYANA 2	VAX 4	VRA 4	VRA 4
4.0 bcd	5.0 b-e	176.8 b-m	0.42 b-m
KAB06F2.8-27	VCB 81013	KAT 56	KAT 56
4.0 bcd	5.0 b-e	182.0 c-n	0.43 c-n
KAB06F2.8-36	CAL 96	NUA 59	NUA 59
4.0 bcd	5.5 c-f	183.0 d-n	0.44 d-n
KAB06F2.8-35	KAB06F2.8-36	Ngakwu-Ngakwu	Ngakwu-Ngakwu
4.0 bcd	5.5 c-f	187.2 d-n	0.45 d-n
KAT 56	KAB06F2.8-35	RWV 2361	RWV 2361
4.0 bcd	5.5 c-f	187.2 d-n	0.45 d-n
KIANGARA	KIANGARA	RW 582	RW 582
4.0 bcd	5.5 c-f	188.2 e-o	0.45 e-o
Local Yield check	Local high Fe check	AGRONOME	AGRONOME

4.0 bcd	5.5 c-f	193.5 f-o	0.46 f-o
Local high Fe check	MAC 74	KAB06F2.8-35	KAB06F2.8-35
4.0 bcd	5.5 c-f	193.5 f-o	0.46 f-o
MAC 42	NDIMIRAKUJA	Local high Fe check	Local high Fe check
4.0 bcd	5.5 c-f	193.5 f-o	0.46 f-o
NDIMIRAKUJA	NUA 99	NDIMIRAKUJA	NDIMIRAKUJA
4.0 bcd	5.5 c-f	193.5 f-o	0.46 f-o
NUA59	RWV 2361	NUA 99	NUA 99
4.0 bcd	5.5 cdef	193.5 f-o	0.46 f-o
NUA 99	RWV 2887	VAX 1	VAX 1
4.0 bcd	5.5 cdef	193.5 f-o	0.46 f-o
RW 582	VAX 1	GLP 2	GLP 2
4.0 bcd	5.5 cdef	198.8 g-o	0.48 g-o
RWV 2359	CODMLB 001	RWV 2359	RWV 2359
4.0 bcd	6.0 def	198.8 g-o	0.48 g-o
RWV 2361	GLP 2	RWV 2887	RWV 2887
4.0 bcd	6.0 def	198.8 g-o	0.48 g-o
VAX 1	ICYANA 2	RWV 3316	RWV 3316
4.0 bcd	6.0 def	198.8 g-o	0.48 g-o
VAX 3	Local Yield check	VAX 3	VAX 3
4.0 bcd	6.0 def	198.8 g-o	0.48 g-o
VAX 5	Ngakwu-Ngakwu	KAB06F2.8-36	KAB06F2.8-36
4.0 bcd	6.0 def	199.8 h-o	0.48 h-o
VRA 4	NUA 69	KIANGARA	KIANGARA
4.0 bcd	6.0 def	199.8 h-o	0.48 h-o
AGRONOME	RUGANDURA	NUA 69	NUA 69
4.5 cde	6.0 def	204.0 i-o	0.49 i-o
CODMLB 001	RWV 2359	ICYANA 2	ICYANA 2
4.5 cde	6.0 def	205.0 j-o	0.50 j-o
LMB 49	RWV 3316	Local yield Check	Local yield Check
4.5 cde	6.0 def	205.0 j-o	0.50 j-o

NUA	69	VAX	3	VAX	5	VAX	5
4.5 cde		6.0 def		205.0 j-o		0.50 j-o	
RW	846	VAX	5	CODMLB	001	CODMLB	001
4.5 cde		6.0 def		216.5 k-o		0.52 k-o	
RWV	2887	LMB	49	RW	846	RW	846
4.5 cde		6.5 ef		220.8 l-o		0.53 l-o	
RUGANDURA		RWV	1129	LMB	49	LMB	49
5.0 de		6.5 ef		221.8 m-o		0.53 m-o	
RWV	1129	RW	846	RUGANDURA		RUGANDURA	
5.0 e		7.0 f		234.2 n-o		0.56 n-o	

NB: Means with the same letters are not statistically different

Appendix 3. Means separation for yield parameters first and second screening

First screening			Second Screening
No of pods/plant	Weight of seed (gr)	100 seed weight (seed size) (gr)	100 seed weight (seed size) (gr)
KAT 69 1.0 a	KAT 69 2.4 a	KAT 69 20.0 a	KAT 69 20.0 a
RWV 3316 4.5 ab	RWV 3316 4.1 ab	RWV 2070 21.1 b	RWV 2070 21.1 ab
CAL 96 6.0 abc	Masindi Yellow 5.7 abc	DOR 500 21.8 c	DOR 500 21.8 bc
Masindi Yellow 6.0 abc	KAB06F2.8-12 6.5 a-d	RWV 3316 22.5 d	RWV 3316 22.5 bcd
Local High Fe Check 6.0 abc	KIVUZU 7.0 a-e	MAHARAJI SOJA 22.9 de	MAHARAJI SOJA 22.9 cde
KAB06F8.8-35 6.2 abc	CAL 96 7.1 a-e	NGWINxCAB2 23.3 ef	NGWINxCAB2 23.3 de
KAB06F2.8-12 7.0 a-d	Local High Fe check 7.9 a-f	ROBA 1 23.9 fg	ROBA 1 23.9 def
Local yield check 7.5 a-e	KAB06F2.8-36 8.2 a-f	VAX 1 23.9 fg	VAX 1 23.9 def
ROBA 1 7.5 a-e	KAB06F8.8-35 8.7 a-g	MIB 456 24.3 gh	MIB 456 24.3 efg
RWV 2154 7.5 a-e	Ngwaku-Ngwaku 8.9 a-g	Gitanga 1 24.8 hi	Gitanga 1 24.8 fgh
USDK-CBB-15 7.5 a-e	USDK-CBB-15 9.3 a-h	NABE 3 24.9 hi	NABE 3 24.9 f-i
KAB06F2.8-36 7.9 a-e	RW 582 9.6 a-i	VAX 5 25.1 ij	VAX 5 25.1 f-j
MAC 74	Local Yield check	MCM 2001	MCM 2001

8.0 a-e		9.7 a-i		25.6 jk		25.6 g-k	
SMC	16	AFR	708	RWR	719	RWR	719
8.0 a-e		10.0 a-j		26.2 kl		26.2 h-l	
KAT	31	KAT	31	ACC	714	ACC	714
8.5 b-f		10.1 a-k		26.3 lm		26.3 i-m	
KAT	56	RWR	2154	VAX	4	VAX	4
8.5 b-f		10.2 a-k		26.4 lm		26.4 j-m	
Ngwaku-Ngwaku		SMC	16	RW	805	RW	805
8.5 b-f		10.3 a-l		26.9 mn		26.9 k-m	
MAC	42	NUA	59	RW	846	RW	846
8.5 b-f		10.4 a-l		27.1 no		27.1 lm	
CAB	2	RWV	2070	NUA	59	NUA	59
9.0 b-g		11.0 a-l		27.6 o		27.6 lm	
RWV	2887	ROBA	1	SMC	21	SMC	21
9.5 b-h		11.2 a-l		27.7 o		27.7 m	
MONTALCAM		MAC	42	SMC	17	SMC	17
9.5 b-h		11.2 a-l		29.4 p		29.4 n	
HM	21-7	RWV	2887	RUGANDURA		RUGANDURA	
10.0 b-i		11.5 a-l		29.6 p		29.6 no	
AFR	708	MONTALCAM		VAX	2	VAX	2
10.3 b-i		11.9 a-l		29.6 p		29.6 no	
CODMLB	001	KAT	39	KAT	56	KAT	56
10.5 b-i		12.1 a-l		30.0 pq		30.0 nop	
RWV	2359	HM	21-7	RW	1180	RW	1180
10.5 b-i		12.2 a-m		30.4 qr		30.4 n-q	
SMC	18	RWV	2245	SMC	18	SMC	18
10.5 b-i		12.3 a-m		30.4 qr		30.4 n-p	
CAL	143	CODMLB	001	RW	184	RW	184
11.0 b-j		12.4 a-n		30.9 rs		30.9 o-r	
NUA	45	KAT	56	VAX	3	VAX	3
11.0 b-j		12.4 a-n		30.9 rs		30.9 o-r	

RWR 3006	CODMLB 003	KAB06F8.8-35	KAB06F8.8-35
11.0 b-j	12.4 a-n	31.1 s	31.1 p-s
SMC	NUA 99	RWV 3006	RWV 3006
11.0 b-j	12.5 a-o	31.4 st	31.4 p-t
RW 582	CAB 2	VRA 4	VRA 4
11.5 b-k	12.5 a-o	31.5 st	31.5 q-t
RWV 2361	SMC 18	AGRONOME	AGRONOME
11.5 b-k	12.6 a-p	31.9 tu	31.9 rst
CODMLB 003	CAL 143	Masindi Yellow	Masindi Yellow
12.0 c-l	12.9 a-q	32.0 tuv	32.0 rst
GLP 2	SMC 21	Nain de Kyondo	Nain De Kyando
12.0 c-l	13.2 a-r	32.2 uv	32.2 rst
NUA 99	RWV 2359	JESCA	JESCA
12.0 c-l	13.6 b-s	32.4 uv	32.4 stu
RW 547	SMC 17	GARUKURARE	GARUKURARE
12.0 c-l	13.9 b-s	32.6 v	32.6 tu
RWV 1129	VAX 5	NUV 219-1	NUV 219-1
12.0 c-l	14.8 b-t	33.8 w	33.8 uv
KAT 39	RW 184	RW 547	RW 547
12.5 c-m	15.1 b-u	33.8 w	33.8 uv
NGWINXCAB2	GLP 2	AFR 708	AFR 708
12.5 c-m	15.4 c-u	34.5 x	34.5 vw
RWR 719	NUA 45	KIVUZU	KIVUZO
12.5 c-m	15.6 c-u	35.5 y	35.5 wx
GASIRIDA	NGWINxCAB2	LMB 49	LMB 49
13.0 c-m	16.1 c-u	36.2 z	36.2 xy
KIANGARA	KAB062.8-27	SMC 16	SMC 16
13.0 c-m	16.2 c-u	36.4 za	36.4 xyz
SMC 21	RWV 2361	CAB 2	CAB 2
13.0 c-m	16.5 c-v	36.5 za	36.5 xyz
VAX 1	MCM 2001	DECELAYA 1	DECELAYA 1

13.0 c-m	16.6 c-v	36.5 za	36.5 xyz
VAX 5	RW 547	RWV 2887	RWV 2887
13.0 c-m	16.9 d-v	36.5 za	36.5 xyz
JESCA	MIB 456	CAL 143	CAL 143
13.5 c-n	16.9 d-v	37.0 ab	37.0 yzA
KAB062.8-27	Gitanga 1	RWV 2361	RWV 2361
13.5 d-n	17.2 d-v	37.4 bc	37.4 yzA
KIVUZO	MAHARAJI SOJA	NDIMIRACUJA	NDIMIRACUJA
13.5 d-n	17.2 d-v	37.6 bc	37.6 yzA
NABE 3	RWV 3006	CODMLB 001	CODMLB 001
13.5 d-n	17.3 d-v	37.8 cd	37.8 zA
NUA 69	RW 846	KAB06F2.8-36	KAB06F2.8-36
13.5 d-n	17.8 e-v	38.4 d	38.4 AB
RW 1180	MAC 74	ICYANA 2	ICYANA 2
13.5 d-n	18.0 e-v	39.6 e	39.6 BC
VRA 4	RWR 719	RWV 2245	RWV 2245
13.5 d-n	18.0 e-v	40.0 ef	40.0 C
MCM 2001	VAX 1	RW 582	RW 582
14.0 d-o	18.4 f-v	40.1 fg	40.6 C
RW 805	AGRONIME	RWV 2359	RWV 2359
14.0 d-o	18.8 f-v	40.6 fg	40.8 C
RWV 2070	VAX 4	MCB 49-89A	MCB 49-89A
14.0 d-o	18.9 f-v	40.8 g	40.8 C
VAX 4	NABE 3	GLP 2	GLP 2
14.5 e-o	19.5 g-w	42.4 h	42.4 D
AGRONOME	NUA 69	KAB062.8-27	KAB062.8-27
14.5 e-o	19.6 g-w	42.8 hi	42.8 D
MIB 456	RWV 1129	VCB 81013	VCB 81013
15.5 f-o	20.3 h-w	43.3 ij	43.3 DE
Nain de Kyando	VRA 4	HM 21-7	HM 21-7
15.5 f-o	20.3 h-w	43.6 j	43.6 DEF

NUV 219-1	RWV 1180	CAL 96	CAL 96
15.5 f-o	20.5 i-w	44.3 k	44.3 EFG
RW 184	DOR 500	GASIRIDA	GASIRIDA
15.5 f-o	20.8 j-w	44.3 k	44.3 EFG
RWV 2245	NUV 219-1	NUA 69	NUA 69
15.5 f-o	21.1 k-w	44.4 k	44.4 EFG
MCB 49-89A	VAX 3	USDK-CBB-15	USDK-CBB-15
16.0 g-o	21.3 l-x	44.4 k	44.4 EFG
MAHARAJI SOJA	JESCA	MAC 44	MAC 42
16.5 h-o	23.1 m-x	44.8 k	44.8 FG
VAX 2	MCB 49-89A	RWR 1129	RWV 1129
16.5 h-o	23.4 n-y	45.5 l	45.5 GH
VAX 3	GASIRIDA	NUA 99	NUA 99
16.5 h-o	23.5 o-y	45.6 l	45.6 GH
Gitanga 1	MAC 44	Ngwaku-Ngwaku	Ngwaku-Ngwaku
17.0 i-o	23.5 o-y	46.4 m	46.4 H
NDIMIRACUJA	RW 805	CODMLB 003	CODMLB 003
17.0 i-o	23.6 p-y	46.8 m	46.8 HI
RW 846	VAX 2	NUA 45	NUA 45
17.7 j-o	23.7 q-y	48.2 n	48.2 I
LMB 49	ACC 714	MONTALCAM	MONTALCAM
18.5 k-o	23.9 r-y	50.5 o	50.4 J
DECELAYA 1	Nain de Kyando	KAB06F2.8-12	KAB06F2.8-12
19.0 l-o	24.3 s-y	50.6 o	50.6 J
GARUKURARE	KIANGARA	RWR 2154	RWR 2154
19.0 l-o	24.6 s-z	53.3 p	53.3 K
DOR 500	NDIMIRACUJA	KAT 39	KAT 39
19.5 m-o	25.7 t-z	53.3 p	53.3 K
RUGANDURA	LMB 49	Local High Fe	MAC 44
19.5 m-o	26.1 u-z	Check 53.6 p	54.7 K
VCB 81013	ICYANA	MAC 44	Local High Fe

9.5 m-o		27.5 v-z		54.7 q		check 56.4 L	
Zebra		DECELAYA	1	KAT	31	Local yield Check	
19.5 m-o		30.0 w-z		57.1 r		56.5 L	
ICYANA	2	Zebra		Zebra		KAT	31
20.5 n-o		30.3 w-z		57.3 r		57.1 L	
ACC	714	VCB	81013	MAC	74	Zebra	
21.0 o		32.3 x-z		57.5 r		27.3 L	

NB: Means with the same letters are not statistically different